MEDICAL SCIENCE

FOR EVERYDAY USE

BY

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TO MY WIFE

ALICE SPRINGFIELD WARREN

PREFACE.

For several years the Boston *Sunday Herald* contained articles on subjects of current medical interest which I had written in an attempt to present in everyday speech the advances and accomplishments of medical science. Encouraged by the interest of my readers, some medical men among them, and by requests for the articles in a more permanent form, I have revised, rewritten and collected in this volume those of the articles which seemed of most value.

I am indebted to the Boston Publishing Company, through Mr. R. L. O'Brien, editor, for permission to reproduce in whole or part my contributions to the *Sunday Herald.* I also desire to acknowledge the interest and care of the publishers, Messrs. Lea & Febiger.

S. W.

Needham, Massachusetts,
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MEDICAL SCIENCE FOR EVERYDAY USE.

PART I.

GENERAL TOPICS.

CHAPTER I.

THE GROWTH OF CHILDREN.

Is that youngster of yours that is half a head taller than most of his playmates going to be a six-footer and be good football material when he grows up, or will he get tired growing after a while and let the others catch up to him? That is one of the things that doctors have been finding out about lately, in trying to discover how fast and how big children ought to grow.

And is it true that Margaret has grown as much in the three summer months of out-door life as in the nine of school? That question, too, has been answered.

The data by which these and other points have been cleared up now form a very respectable mass and represent the growth of boys and girls in the different sections of the country. Perhaps the most important studies are those by Dr. Bowditch and more recently Dr. Porter on thousands of Boston school children, by Dr. Porter on over 34,000 St. Louis children and by Dr. Baldwin on thousands of

boys and girls in Iowa. Dr. Terman in California has carried out for years careful studies on not only the physical but mental development of great numbers of the so-called "superior children."

Of course similar studies have been made abroad, but owing to racial variations cannot be applied directly to conditions here, although the findings are substantially in agreement with the American results.

It is just as well to know a little about the babies before we begin to talk about the children. The most fashionable weight for new-born babies appears to be 8 pounds. Though rarely they may be as light as 4 pounds and as heavy as 14, there are more 8-pounders born than any other weight. So the next time a proud father asks you to guess the weight of the new arrival, 8 pounds will be your best bet. There is not very much variation in length, a shade over 20 inches being the average.

Once in the world, two principal things influence the baby's growth—the food he gets and the diseases he has. Artificially-fed babies as a rule gain less for at least the first few months. Chronic illnesses, such as prolonged diarrhea, bronchitis and running ears retard growth considerably. But with any decent chance, a boy will double his weight by the time he is seven months old, and a girl when she is eight months. With babies the season does not make much difference—they are growing on their own schedule, and a baby born in January, other things being equal, grows at about the same rate as one born in July.

These studies, and others in various American cities when compared with those made in France and Germany, show that even before the World War the French and German infants were lighter and shorter than the American.

In the first six years after birth, whether in Boston or Sioux City, the average American youngster doubles his height and increases four times in weight. If he kept on growing at this rate, by the time he was eighteen years

he would be 10 feet tall and weigh a quarter of a ton. In view of the present prices of food and clothes, it is just as well that he slows down a little. Through these six years, and up until about eleven years old, the boys are generally not only taller and heavier than the girls of the same age, but weigh more in proportion to their height. This is chiefly because they are deeper chested and better developed muscularly.

During these years a change in rate of growth varying with the seasons appears in both boys and girls, and becomes most marked during the school age. From a series of measurements of nine-year-old Boston school boys, Dr. Porter found that they gained only $\frac{3}{4}$ pound from January to June, and $3\frac{3}{4}$ pounds from June to November. This was regardless of the months in which they were born. On extending his observations, he was able to work out the average monthly gain for school children—except that he had to lump the months of June, July and August, during which the schools were closed.

His figures, expressed in pounds, are as follows: January, gain of 0.18; February, 0.47; March, 0.22; April, loss of 0.16; May, gain of 0.05; June-July-August, 2.23; September, 0.96; October, 0.61; November, 0.63; December, 0.98. That is, no matter whether a child is born in February or August, he gains very slowly in the winter and spring, even losing a little weight in April, and then shoots ahead in the summer and fall. The outdoor life in these last two seasons, and the rarity of colds and other diseases as compared with winter and spring, are the most likely explanations.

All this time, the boys have been taller and heavier than the girls. But at the age of eleven or twelve years, the girls begin to grow very rapidly, and outdistance the boys for a couple of years. At fourteen or fifteen years, boys have their spurt of rapid growing and regain their lead. However, boys keep a greater chest expansion, length and width of face and muscular strength throughout. When

children are over 5 feet tall, girls are heavier than boys in proportion to their height, though below this the boys have the lead.

What are the chances that two brothers or two sisters will be about the same build when they grow up? Pretty fair, according to the results of Baldwin's studies on thousands of Iowa children, but the resemblance between their "growth curves," or rate of increase in height and weight, is even more striking. Those for the boys in any one family are usually very similar, and those of sisters are much alike. That is, if John's older brother grew very fast when he was seven years and then barely held his own for a year or two, the chances are that John will do the same thing when he gets to be the same age.

There is apparently some correlation between physical and mental development. In a study of over 34,000 school children in St. Louis, Porter found that the brighter children were taller, heavier and more deep-chested than the average. It is also well known that a condition such as diseased adenoids, which stunts physical growth, causes mental slowing as well. However, though in many cases a child shows great mental and physical improvement following removal of diseased tonsils and adenoids, this operation cannot be regarded as a sure cure for stupidity or retarded development.

The practical bearing of all this is that if a child is below par, he probably has some trouble which should be found and corrected. But first we have to have a standard. Weight alone is a poor one, because it fluctuates a great deal, often for little reason. Height is more constant, but height alone is not worth much. We have all seen tall, frail youngsters that looked ready to wilt, and short, chunky ones husky enough to eat nails.

There are two requisites for any practicable standard. First, it must be readily applied to large numbers of children. Secondly, it must give definite and reliable information.

The condition of the youngsters is what we are interested in. We do not want to fatten them as so many hogs or steers, but we know that if their weight gets too low through undernourishment, they are an easy prey to disease. It happens that the relation of weight to height of children, when due allowance is made for their age, gives a very serviceable index to their nutrition and through this to their ability to withstand disease. Both weight and height are figures easily and accurately obtained, and but slight allowance has to be made for errors due to the personal equation of the examiners. Moreover, measurements of this type can be made much more frequently than a thorough-going physical examination. In rapidly-growing children frequent examinations are of no slight value. A sudden slowing in rate of growth, for example, usually points to trouble.

The common standard is the relation of the weight of a child to its height as compared with other children of the same age. Thousands of Boston school children were weighed and measured by Dr. Bowditch of the Harvard Medical School years ago. The standards which he set have been modified to some extent by many other careful surveys made in different parts of this country and abroad, but remain essentially unchanged.

So when the school nurse tells you Johnny is under weight, it means that he weighs less than the average of several thousand other children of the same age and height. Now it is perfectly obvious that all eight-year-olds are not going to weigh just the same, any more than they all look alike.

Instead of saying that a pound out of the way is too much or too little, a variation of 10 per cent is generally regarded as permissible. That is, a boy weighing 54 pounds is not considered overweight if the standard for his age and height is 50 pounds. The reasonableness of this latitude is plain when we remember that our standards are not made from picked, healthy children, but the average of many children,

the great bulk of whom were healthy, but varying widely in build and physical condition.

Some investigators believe that 10 per cent above and below average is too wide a range. They feel it is better to regard any child over 7 per cent below the standard as under-nourished unless he can be proved otherwise. Here the danger is that by too frequent crying of "wolf," parents will be led not to heed real danger signals.

If a child is under-nourished he is made more susceptible to disease, if indeed his loss of weight is not due to disease already existing. If we can head off trouble, so much the better. Under-nourished children are peculiarly susceptible to tuberculosis, among other diseases.

The ratio of weight to height is about the best single guide, as it gives an index to the general solidity and robustness of the body. The weight in pounds divided by the height in inches gives what is known as the weight-height index. Thus, if a girl is 50 inches tall and weighs 60 pounds, her weight-height index is 1.2. This index rises steadily with age up to the time of maturity. For five-year-old boys it averages 0.91, and rises steadily until at seventeen years the average is 2.03.

In interpreting the measurements of childen, care must be taken not to confuse the average with the normal. There is a considerable range both above and below the average which may be normal for any given child. Ordinarily, as we have said, a range of 10 per cent above or below is considered normal. But if a child has been consistently 5 per cent above the average and then drops to 5 per cent below the next time he is measured, something is wrong.

The weight-height index is a good starting point for observation, but the surest way to check up progress is to keep track of the annual increase in weight and height at the different ages.

Boys.

	Weight increase, pounds.	Height increase, inches.
6	4.00	2.0
7	4.75	2.0
8	5.25	2.0
9	6.00	2.0
10	5.00	1.7
11	6.50	1.8
12	8.00	2.0
13	10.00	2.5
14	12.50	2.7
15	13.75	2.7
16	6.50	1.2

Girls.

	Weight increase, pounds.	Height increase, inches.
6	4.00	2.00
7	4.50	2.00
8	5.00	1.75
9	5.25	2.25
10	6.50	2.00
11	9.50	2.50
12	10.50	2.00
13	9.50	2.00
14	7.50	1.25
15	6.00	0.75
16	3.50	0.50

A little extra care and oversight on the part of the parents can often convert an under-nourished youngster into a healthy one. The average parents, however, so long as a child is apparently well, have no reason to suspect that anything is wrong. It is to give them warning in time to head off any possible trouble that the school doctors and nurses weigh and measure the youngsters and compare the results with the normal standards.

CHAPTER II.

WORK AND EXERCISE.

Work is essential to life, at least for most of us. So it is only natural that, along with the study of the various diseases and of the function and structure of the different parts of our bodies, the muscles have come in for a large share of attention. An English physiologist, Dr. A. V. Hill, has so far refined and added to our knowledge of the way our muscles work that now the story seems to be complete. Curiously enough, much of Dr. Hill's experimental material was provided for him by men who had no idea they were helping scientific work, but were intent on breaking athletic records. The various world records for different sports represent the greatest efforts to which humans can drive their muscles, and form a complete body of facts bearing on the physiology of exercise.

When a muscle contracts, heat and lactic acid, the same acid as in sour milk, are produced and work is done—something lifted or shoved. Even when we merely show off our muscle, the muscles on the front of the arm pull against those on the back, doing work thereby, and if we keep up the contraction long enough and hard enough we begin to perspire from the heat produced.

A muscle can be compared roughly to a steam engine—not the kind we are used to on locomotives or steam shovels, where the waste steam is allowed to blow off, but those on steamships, where the steam is condensed to water and used over again. Only instead of water we have glycogen, or animal starch. This is the form in which the starches and sugars from the food are stored in the body. Owing to this

formation of glycogen from starches and sugars, they are the best foods to supply energy.

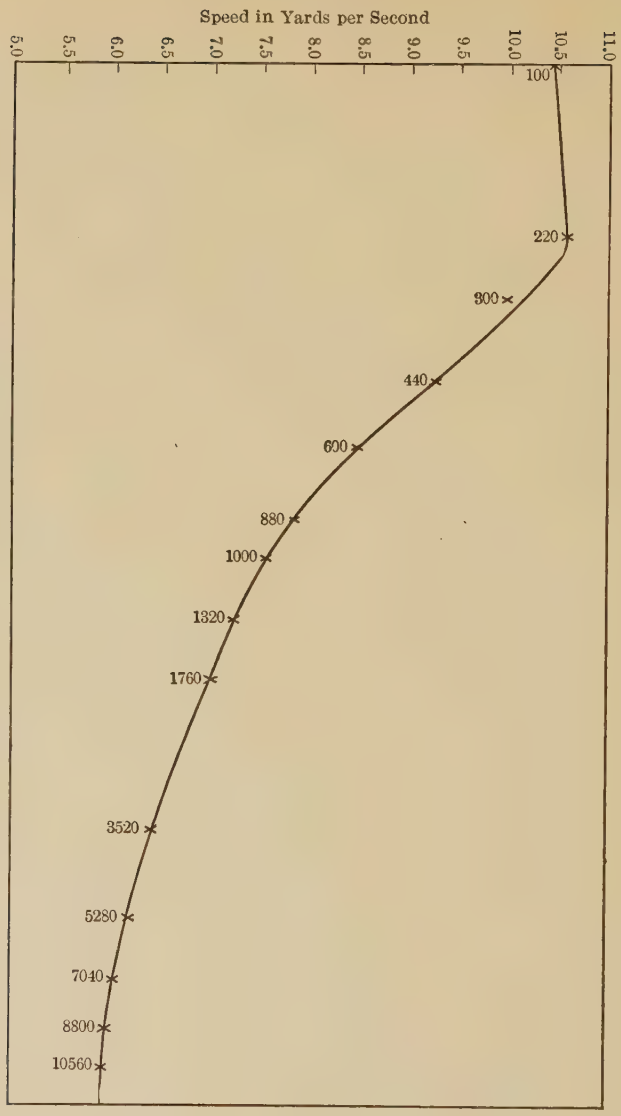
During muscular work, some of the glycogen present is changed to lactic acid. In this change, heat is given off and the muscle contracts. Then when recovery takes place, the lactic acid is changed back to glycogen. The presence of oxygen is essential for both of these reactions. One great advantage the muscle has over the steam engine is that it derives the energy needed for the change from lactic acid to glycogen from the lactic acid itself. One-fifth of the acid is used up, changed to carbon dioxide and water, and the remaining four-fifths are thereby changed to glycogen.

Thus a fatigued muscle is one rich in lactic acid; a rested one is rich in glycogen. The lactic acid is not free in the muscle however, except in very small amounts. We are very sensitive to any change in the reaction of our bodies, and if the amount of lactic acid produced during a day's work at manual labor or even three sets of tennis were present as free acid, we would be dead. As the lactic acid is formed it combines with the somewhat alkaline sodium-protein combination in the muscle, and to a less degree with the phosphates and carbonates of the blood. In this way the lactic acid can be handled without danger to the body.

The quantity of lactic acid produced is hard to realize. An athlete running as fast as he can forms 1 ounce every eight seconds. A great deal of oxygen is needed to change this lactic acid back to glycogen, far more than can be breathed in during strenuous work. It is for this reason we breathe deeply after exercise.

The oxygen used by the body is an accurate index of the energy output. One liter (about 1 quart) of oxygen consumed forms enough energy, if it could be applied without loss, to raise 1 ton 7 feet into the air.

When exercise is over, the intake of oxygen stays above normal until the lactic acid which has accumulated in the


Speed in Yards per Second
5.0
5.5
6.0
6.5
7.0
7.5
8.0
8.5
9.0
9.5
10.0
10.5
11.0
100
220
300
440
600
880
1000
1320
1760
3520
5280
7040
8800
10560
Distance in yards (logarithmic scale), world's records for running.

body due to lack of oxygen has been made over into glycogen. This oxygen intake above the normal level, the level when we are at rest, is a measure of the "oxygen debt" and indicates the amount of lactic acid which has piled up due to lack of oxygen and is changed back to glycogen during the recovery period.

In a 100-yard dash the oxygen debt may be as great as the amount of oxygen an average man would breathe in one-quarter of an hour. In running, for example, the lactic acid is constantly being formed in large amounts, and the rate of formation increases with the speed. Consequently, the need for oxygen increases with the speed. However, the oxygen intake soon reaches its limit; we can breathe only so fast and so deep. Once the oxygen intake is less than the oxygen requirement, lactic acid accumulates and fatigue follows more or less rapidly—more rapidly in untrained men than trained.

Ordinarily we do not get tired—muscularly at least. The intake and need for oxygen are balanced and the lactic acid is taken care of as it forms. With moderately hard exercise we feel "all in" after a while. We pant and feel breathless—we have an oxygen debt that must be made up. This oxygen debt varies greatly, being high or low as the amount of oxygen we breathe in falls more or less short of the requirements. It also varies in different individuals, that is, some can stand greater oxygen debts than others. The trained athlete, for example, can stand a very heavy oxygen debt.

This oxygen debt or the concentration of lactic acid a man can stand can be regarded as his capital and his oxygen intake as his income. The oxygen intake, of course, is governed by the efficiency of the lungs and heart. An athlete in a race, where he makes every effort, will finish with his income all used up and his capital drawn down to the limit.

In the accompanying chart the various world's records for

running are plotted, showing the distance run and the speed per second. The curve is practically level, at an average speed of about 10½ yards a second, from the 100-yard dash to the 220. When we come to the 300-yard run, the speed has dropped to 10 yards a second. The 1000-yard record figures out to 7½ yards a second. The rate thus drops rapidly from the 100-yard to the 1760-yard run, and from there to long distances decreases only a little.

This can be interpreted in terms of our oxygen-lactic acid theory. Up to distance of 220 yards a well-trained athlete can run using up both capital and income at the most rapid rate and so getting constant speed. But at 300 yards and beyond, he has not enough capital—capacity for taking care of lactic acid—and has to depend on his income alone to make up the deficiency. This of course pulls down his speed by slowing the recovery of his muscles. And the longer the distance run, the less will his capital help to maintain his average speed. At about 5000 yard distances the rate of speed steadies down to a level approximately that made possible by oxygen intake alone, and there is little decrease in the average rate even up to marathon distances.

When the best trained athletes run short distances, where the reserves of the body can be used to best advantage, they can make an average speed of a little under 11 yards a second, but in long runs where the reserves are not great enough in proportion to the distance to have any effect on the speed, the oxygen intake is sufficient only for just under 6 yards a second.

Every one has had the experience of being able to sprint one-half block for a car at a rate that would utterly exhaust him if kept up for two or three blocks. It is the same principle again.

It is not enough, however, to get the oxygen into the body. It must be carried by the blood to the muscles where it is needed. Several factors aid in increasing the blood flow to the working muscles. The output of the heart increases

in trained individuals six to nine fold. At the same time the walls of the large bloodvessels in the abdomen contract and those of the vessels of the skin and working muscles relax so that the blood is shunted toward the working muscles. As a result, the blood flow through an active muscle is six to eight times greater than through the same muscle at rest.

The blood itself is better off for exercise. The red blood cells, which do most of the oxygen-carrying for the blood, are not very long lived—about thirty to forty days. As the average number in a small drop of blood is over 4,500,000, the bone-marrow, which has the job of making the red blood cells, must be constantly at work turning out new ones to take the place of those which have worn out. Under normal circumstances this blood cell formation keeps exact pace with destruction. Severe exercise causes an increase in blood destruction, presumably of the older, nearly worn out cells, but if the person is in good trim, the bone-marrow will readily respond to the need for fresh red cells.

The recovery process is a most important one and until recently only vaguely understood. Recovery depends on getting oxygen enough to change the lactic acid formed back to glycogen.

The muscle thus acts not unlike a storage battery. When fully charged it has an ample supply of oxygen and practically no lactic acid. When run down there is an excess of lactic acid. To recharge a supply of oxygen is necessary, just as an exhausted storage battery needs to receive an electric current.

But the muscle has one advantage over the storage battery. The more it is used the better it is. A muscle which is frequently in use recovers much more readily than one unaccustomed to exercise; or to put it the other way around, does not tire as easily. This is largely because a muscle often used has a better blood supply than one seldom used.

The speed of recovery depends not only on the amount

of oxygen we breathe in, but also on the amount the circulating blood can carry to the muscle where the excess of lactic acid is. So the better blood supply a muscle has, the more easily it recovers, and the less strain the heart is under to force sufficient oxygen-carrying blood to it.

In running the blood is easily supplied to the muscles. Gravity helps the heart, the leg muscles have many bloodvessels, and the muscles move rapidly and are never rigid. In rowing the strain on the heart is much greater, since the bloodvessels of the arm are less abundant, the muscles move more slowly, and are rigid for considerable periods.

A familiar experience to all who have done any hard work is the coming of the "second wind." The change from distressing shortness of breath and exhaustion to easy and comfortable breathing and a feeling of being able to keep up the exercise for hours is very striking. This change is probably due to a diminished acidity of the blood, which in turn is brought about by greater mechanical efficiency of the muscles, as they "hit their stride" and a better blood supply is brought to them.

An aspect of exercise which must be considered is its possible bad effect. Overstrain of the heart is the ill most commonly charged to exercise. There is no doubt that, if the nutrition of the heart has been impaired by a recent severe disease or long sedentary life, sudden violent exertion so injures the heart that it is barely able to pump enough blood to maintain the body at rest. When the added demand of exertion is thrown on the injured organ, it cannot meet the demand and the results may be disastrous.

CHAPTER III.

DIET AND OBESITY.

I WENT to dinner the other night with an old friend I had not seen for some years. We sat down, and I ordered a good meal from oysters to ice cream. He ordered crackers and milk, explaining that he was on a diet. Then I looked at him carefully. He did not look any too well, whether in spite of or because of his diet, I do not know. I did not ask him much about it, but learned that milk, crackers, eggs and oranges were all he could eat.

Many of you who are reading this have been "on a diet" one time or another. Most of us regard a diet as a small list of things we may eat and a very large list of things we cannot. This impression is in part due to the way we make a diet.

Stop and think a moment, and you will probably find you have a home-made diet of your own. Perhaps you had a mean night after eating lobster once. Thereafter lobster is on the black-list. From time to time other articles may be made taboo. Perhaps tomatoes, or asparagus, or cabbage, until the list of "do not eat" foods becomes of very respectable length. I cannot eat raspberries—they made me sick once, and I do not care much about them anyway, so I am on a raspberry-free diet.

One of my medical friends told me of a patient who came to him on a milk and buttermilk diet. This man had had persistent stomach trouble for some years. He stopped smoking and drinking coffee, but with no effect on his stomach. He gave up candy and sweets. Then he went to a doctor who told him not to eat meat; even this did not help him. First one thing and then another joined the

forbidden list until milk and buttermilk alone were left. Still his stomach trouble refused to be starved out. Luckily, the story has a happy ending. Once the man began to eat the three square meals a day my friend told him to tackle, the indigestion vanished. The original cause of his trouble had cleared up long ago, and it was his diet that kept him sick.

Most of us have the wrong idea of diets. We have been so often told, "Do not eat red meat," or "Stop coffee and tobacco," that we think of diets as a list of forbidden foods. Instead of that, a diet should be a list, and a long one, of things to be eaten.

Boil down the varied diets—from the fanatical starvation treatment Upton Sinclair fights for to the coldly scientific "high-fat" diet of Drs. Newburgh and Marsh—and when all the sputtering and bubbling is over and the steam has blown away, you have just this: an attempt to give the body just what it needs to keep running in good order, and to avoid giving it excessive amounts of waste to dispose of.

A diet should be based on a careful study of just what the person who is to eat it needs in order to keep his body running in good shape, while avoiding excessive formation of waste products.

Take a disease in which a careful diet is absolutely essential to the well-being and even life of the patient—diabetes. There the body has difficulty in disposing of sugar. Feed foods rich in sugar, and the body becomes swamped with it. The starchy foods are changed to sugar by the digestive juices. The sugar intake must be kept down to that which the body can dispose of. Since starchy foods make up the largest part of our ordinary menu, whether it be breakfast, dinner or supper, the problem of how to cut them down is not easy.

After many failures, a diet was finally worked out that supplied the body most of the substances it required and yet did not clog it with too much sugar. This has to be a

fairly rigid diet and, according to what most of us think, would not have much variety to it. But read over this list of vegetables, any of which may be eaten: lettuce, cucumbers, spinach, asparagus, rhubarb, sauerkraut, beet greens, dandelion greens, swiss chard, celery, mushrooms, tomatoes, brussels sprouts, okra, cauliflower, egg plant, cabbage, radishes, string beans and artichokes—not much lack of variety to that, is there?

Anyone who has been on a diet, and a lot of us who have not, have heard a good deal about calories. A calorie is hardly worth the fuss made over it. It is the amount of heat required to raise 1 gram (about 16 drops) of water 1° C. One thousand calories make 1 Calorie. It is in these large calories that fuel values of our various foods are calculated. When we say ½ dozen oysters give 49 calories, we mean that if the amount of carbohydrate, protein and fat in the oysters was completely burned, the resulting heat would raise 49 liters of water 1° C.

Calories are all right so far as regulation of the fuel requirements of the body goes. But beyond that they fail woefully. A man would be about as badly off if he got his total number of needed calories, and some to spare, from cottonseed oil, as he would if he were totally starved.

A diet calls for more than a regimen which supplies the number of calories the body needs. We have been and are being educated to revere the calorie. Far be it from me to smash its feet of clay, but it is just as well for us to know that they are clay. In addition to counting our calories, we must keep track of the sources from which they come.

Our bodies need other things besides calories. They need lime and salt, protein to repair the wear and tear of living and working, the various vitamins, and a certain amount of roughage.

In these days of white flour and refined sugar, when vitamins and iron are consciously sought for with the aid of numerous advertisements, it is only too easy to slip into a

potato, bread, jam, tea habit. They are foods easy to buy, easy to cook, palatable, and satisfy the hunger. But man cannot live by starch alone, and he gets into trouble if he tries to. We have to remember that our bodies need the building material and roughage obtained from milk, vegetables and meats, as well as the fuel which starchy foods supply.

The starchy foods, carbohydrates, provide immediate energy, the fats are sort of a reserve fuel supply, and the proteins are tissue builders, replacing the daily loss in the body from wear and tear, and providing for growth.

Protein is the class of food that has had most attention paid to it, attention that is well deserved. For it is out of proteins that we are mostly made. Proteins are very complex chemical compounds built up of rather loosely-bound amino-acids. These amino-acids are the bricks out of which the body is built. Combined in various ways to form different proteins, they form the essential part of the various tissues of the body. The body can and does build proteins from amino-acids, but it cannot make the amino-acids. These have to be obtained from the protein in the food.

Since protein is the most expensive and difficult food to produce, much effort has been directed to finding out how much protein is needed and why it is needed. These various experiments, based chiefly on the output of nitrogen in the urine when various diets are being fed, show that about 10 grams of protein food daily is enough to replace the body proteins destroyed by the general wear and tear of life. An adult cannot store protein as he does fat or carbohydrates, in a form readily mobilized for use, though on the ordinary diet there is a surplus of protein in the body. The best he can do is to split off the nitrogen from the amino-acids or the proteins and use the residue as carbohydrate food. When actively growing, however, the increasing number and size of the body cells enables protein storage to go on. A growing pig, for example, can store 30 per cent of the protein fed.

Theoretically, there is no reason to eat any more than

minute amounts of protein, getting most of the needed calories of the diet from fat and carbohydrate. Yet when the diets which people eat when left to themselves are studied, they are found to contain over ten times as much protein as is necessary, if we can trust to our experiments. Shall we trust instinct or science?

Horace Fletcher, of chewing fame, believed science was right and cut his protein down to the limit. He and his followers seemed to show no evidence of lack of protein. It must be more than coincidence, however, that has caused man to maintain an average daily protein intake of 80 to 100 grams whenever that amount is obtainable, whether he is rich or poor, in Norway or Nicaragua. It is the non-protein elements in the diet that vary widely.

So, whether we need it or not, our natural inclination is to get a fairly large amount of protein. Protein is the hardest food to produce, but some proteins are much easier to produce and are less expensive than others. But are these cheaper forms just as good?

One of the first points to determine is the wastage of the protein in digestion. This is done by estimating the amount of nitrogen in the food and then testing quantitatively for the protein which has passed through the body undigested. Of course one slight source of error is that nitrogen compounds may be excreted into the intestines, but the error is not large enough to make much if any trouble.

The proteins of milk and meat are practically completely digested, unless they have been dried or much overcooked. White of egg, which most of us consider the last word in digestibility, is only 58 per cent absorbed when cooked, and when raw and unbeaten is still less digestible. Beans and peas come about midway between milk and egg-white. The small amounts of protein in fruits, such as apples, prunes or strawberries, are almost entirely undigested.

Cooking helps greatly in making plant proteins more digestible, largely by breaking open the cellulose shell which

keeps the digestive juice from the proteins. The peanut, whose protein is highly indigestible, is not improved any by cooking.

Cottonseed oil, used to some extent for frying and the like, has a protein which is digested easily when obtained chemically pure, but which is entirely indigestible otherwise, due to the presence in the oil of another substance which prevents the action of the digestive juices on the protein of the oil.

While these facts hold for feeding pure proteins, they do not hold rigidly on a mixed diet. Any farmer knows his stock get more good out of grain or other concentrated food if fed a certain amount of roughage at the same time. Some recent experiments on feeding hogs show that the digestibility of corn protein increased from 49 to 77 per cent when a fairly large amount of milk was added to the diet.

Once digested, protein has to face still more wastage in meeting the requirements of the body. The digested protein is split into its constituent amino-acids, and those are used or not, according to the need. Some proteins contain amino-acids better suited than others to repair the wear and tear, just as one table in a bargain basement may contain an assortment of shoes all too small, while on the next table they are just right.

The ultimate food value of a protein, allowing for digestibility and for wastage in the body, is known as the biological value. It is in this biological value that proteins vary most. Say you eat a rumpsteak, which is 20 per cent protein. Of this only 95 per cent is digestible, which leaves us with 19 per cent digested protein. About 80 per cent of this absorbed protein consists of amino-acids that can be used for replacement or repair work; the remainder is used for fuel only. So the net protein value of steak is only 15.2 per cent.

Now take the navy bean, with a little more protein than steak—22 per cent. Eighty per cent of its protein is digestible, but only 38 per cent of the absorbed amino-acids are usable. So, although the bean starts with more protein

than the steak, its value as a protein food is only 6.7 per cent against 15.2 per cent.

Using a number of different animals, attempts have been made to calculate the average efficiency of protein foods, taking 100 as the base. The most accurate and careful of these experiments show the proteins of milk to head the list, with a value of 85. Others are lean pork, 74; wheat, 67; potato, 67; corn, 60; navy beans, 38. This rating is of course solely on the protein of the given foods, entirely disregarding any fat or carbohydrate present.

One important fact to keep in mind is that the most valuable proteins of wheat are discarded in the refining process. Consequently, the protein value of white flour is far below that of the whole wheat, and in most comparisons between cereal proteins and meat proteins whole grain has been used to determine the values.

On the average, 43 per cent of the protein in the usual American diet is derived from milled cereals and 9 per cent from beans or peas. Without admixture of milk or meat proteins, the biological value of such foods is very low, particularly for a growing child.

There are many aspects to this problem of the various foodstuffs to use and the amount of them necessary and desirable, though the one that touches most of us is the relation of food to body weight.

Every one may like a fat man, but most fat people spend a good deal of time and money trying to get thin. One of the most fruitful fields for the quacks and patent medicine fakers is in exploiting supposed remedies for obesity. Besides these "remedies," there are all sorts of diet lists, some of articles tabooed, others of articles to be eaten. There are nearly as many styles of exercise, from horseback riding to rolling, as there are fat people. Salt baths, sweat baths, mud baths all have their adherents. The desire for a sylph-like form seems to increase as its attainment becomes more difficult.

Obesity can result from a number of different things. Probably the commonest cause is the same as for many other disturbances of bodily function—underexercise and overeating going hand in hand.

This type of fatness usually tends to come on during and after middle age, as one's activity becomes less.

Another definite variety of fatness is that due to disturbances in function of the ductless glands. Tumors in or near the pituitary gland, which is tucked away in a bony pocket beneath the brain, probably give the most striking degrees of obesity, though such cases are very rare. Extreme fatness due to pituitary disorders usually appears in young people.

Aside from the fat we put on by eating too much and not exercising enough, and fat that is a symptom of trouble in the ductless glands, there is a third and unexplained type of fatness. For some reason there are people who are fat by nature. They do not eat more than other people and their energy output, as far as can be made out by very careful experiments, is about the same as that of thin people.

One possible reason for the increased value fat people of this type seem to get from their food has been brought forward recently. After eating a meal rich in protein or carbohydrate the amount of energy used by the body at rest increases in amount, apparently due to the processes of digestion. This is called technically the specific dynamic action of food, and means that a certain amount of the food eaten is burned up without either serving as fuel for work or being deposited in the form of fat for a reserve food supply.

By careful measurement of the energy output of obese people, at least those whose increased fat cannot be explained by eating too much or glandular disorder, it has been discovered that this specific dynamic action is much less marked in them than in normal or thin persons. They do not use as much of their food in this apparently wasteful process as most of us do, and so even though they do not eat more, they have more surplus food available for storage as fat.

There are a number of tables giving standard weights for various heights and ages. These are not always at hand, however, and a good method of determining normal weight, though not entirely accurate, is the following one. Multiply the number of inches over 5 feet in a person's height by 5.5, add 110, and you have a fair approximation of his normal weight. These weights are possibly a little generous, but when dealing with humans fixed rules are impossible. A leeway of 20 pounds above or below the standard is allowable, even though not advisable.

Thus, if a man is 5 feet 10 inches high, the number of inches above 5 feet is 10. This multiplied by 5.5 is 55, and added to 110 gives 165, the number of pounds he should weigh.

The bony framework of some people is heavier than that of others, and other factors such as race enter in, so a variation of 5 to 10 pounds from the standard is entirely within normal limits.

Fat in the body is a good deal like carbon in an automobile engine. It represents unused fuel. Unlike carbon, it can be used as fuel under certain circumstances. Just as carbon makes it harder for an engine to work, fat throws an extra load on the body. Just as carbon eventually accumulates enough to make serious trouble, an excessive amount of fat may cause serious trouble.

The great weight thrown on the feet causes flat-foot and arch trouble, and the increased load the joints of the legs have to bear may produce arthritis. Acute infections, such as pneumonia, boils or blood-poisoning, generally make far more trouble for fat people than they do for thin ones.

Moreover, evidence is steadily accumulating to show that obesity is one of the important predisposing causes of diabetes. Dr. Elliott P. Joslin, who probably knows more about diabetes than any one else, feels that obesity is a considerable factor in bringing on the disease. So far as I know, most students of diabetes agree with him.

Last, but not least, comes up the question of appearance. For every fat person consulting a doctor on account of loss of health there are a goodly number seeking help because of loss of their figure. It is to this last group that the attractively advertised but rather dubious patent medicines, salt baths and mechanical reducers make their appeal.

Unfortunately there is no royal road from fatness to sylph-like figures. The job of reducing cannot be hurried without harm being done, and rather than trying to run the rapids alone, trusting blindly to fate, it is much safer to take on your doctor as pilot.

Fat cures are particularly trying for children and for old people, and ought not to be attempted unless there is definite need. In any case, do not try to get thin all at once.

The principle of the treatment of obesity by restricting the diet is simple enough. The body needs nitrogen, obtained from protein, to make up for wear and tear and provide for new growth. Consequently a diet is given which will supply plenty of protein, and the carbon-containing portion of the diet is cut down. In this way, the body is provided with all the material it needs for repairs, but rather less than it needs for producing fat, or even for running normally. A diet of this type not only cuts down the food available to form fat, but means the body will have to burn a portion of the stored up fat in order to get enough energy.

Moderate exercise, gradually increasing in strenuousness, is of great value in getting rid of some of the surplus fat.

For a good general reducing diet, keep away from butter, fat meat, oils and fried foods. Cut down on sugar, excessive amounts of fruit and salt. Follow the diet your doctor makes out for you. Meanwhile drink all the water you want, do not try to reduce too fast, and do not be discouraged by the slowness of your progress. Remember it is just as dangerous to be in a hurry to change over the body as it is to be in a hurry at a railroad crossing.

CHAPTER IV.

THE HEALTH OF SCHOOL CHILDREN.

The time was when schools were rightly regarded as hot-beds of disease. But thirty years ago Dr. Samuel H. Durgin, Commissioner of Health of Boston, decided it was time to do something to check the great increase in diphtheria, scarlet fever, measles and other contagious diseases that come at the opening of the schools in the fall. Through his efforts was established in Boston the first systematic medical examination of school children in America.

This examination we have now come to regard as a matter of course and a valuable help in looking after our children, but at that time it was a decided innovation and met with much opposition. But now its value has been so thoroughly proved that hardly a school in the larger towns and cities of the country is without it.

Many defects which have gone unnoticed by the parents have been discovered and means for their correction supplied. Many a parent of a near-sighted youngster or one with adenoids has been surprised and delighted by the improvement both mentally and physically when the unsuspected handicap has been removed.

Besides these direct benefits, everyone interested in growing children has been helped by the statistics gathered in the examination of thousands of children. The examination not only helps the children gone over, but through study and application of results the children of years to come.

Useful as periodic physical examinations are, they are not enough. An inspection once, twice or three times a year is valuable to the child examined, but cannot have much

effect in preventing contagious diseases. Even though his intentions are the best possible, Johnnie cannot save up having the mumps until time for the medical inspection. He is going to get the disease whenever the germs happen to meet him under the right conditions. Consequently, the amount of contagious disease was not reduced as much as had been hoped.

The constant activity and attendance of the school nurse is a great step in advance. She can detect the early symptoms of disease, and so be of great aid in heading off epidemics. Moreover, she knows both children and parents much better than the school physician does. This means she knows and sees the children not only in school, but in play time and at home as well.

Yet, not even the school nurse knows the children as does the teacher. Seeing the same faces for long periods day after day, knowing more or less about the homes from which her pupils come, the teacher is perhaps better fitted than any one except the child's own parents to detect early symptoms of disease. Every school system has made some use of this peculiar opportunity the teacher has to look after the physical well-being of her pupils.

The teachers should be trained to notice and report to the school physician, who then can make careful examination, the commoner symptoms of disease. Perhaps the most important are skin eruptions, paleness, shortness of breath, sore throat or running of the nose.

Though practically every school system has made tacit use of the teacher as a guardian of her pupils' health, so far as I know Minneapolis is the first city to give her a definite place in the system of medical inspection. The way that it is done is simple and adds but little to the teacher's work.

Recently 18 of the grade schools, about one-fifth of the total number in the city, were selected in which to try out the method. The schools, which were chosen to represent

every part of the city, had a total enrollment of nearly 10,000 children. The system was started after the Christmas vacation and kept up until the spring vacation, when the number of communicable diseases begins to drop down.

The school nurse at each school was furnished with diagrams showing the seats in the various rooms of the school. When the teacher noticed a child who appeared to be unwell, she notified the school nurse. After looking over the youngster, if anything was wrong the nurse called in the school physician. If it turned out to be a case of contagious disease, she marked in the space for the child's seat on the diagram the name of the disease and the date.

Likewise if a doctor reported to the board of health a case of contagious disease in a pupil, the report was sent on to the nurse, who entered on her diagram this case in the same way. Then she found out from the teacher the chums of the sick youngster. From her chart she knew the ones who sat nearby. On these children the nurse and teacher kept a specially sharp lookout for early symptoms of disease.

Fore-warned is fore-armed in fighting disease as well as anything else. By means of the Schick test we can pick out the children susceptible to diphtheria and with toxin-antitoxin immunize them against the disease. With the Dick test and scarlet-fever toxin for immunizing purposes we can control scarlet fever. So far as the other contagious diseases are concerned we have to depend largely on quarantine measures. Consequently, the sooner cases are detected, the more effective our control will be. It is from the missed case of disease that others are more apt to be contracted.

Unfortunately most of the common diseases of childhood are highly infectious before they can be readily recognized. For example, the child sick with measles, though only in the sniffling, coughing stage, is just as apt to give it to his mates as he would be with a full-blown rash. Cases are on record where a child sick with measles has infected others four days before the measles rash appeared on his skin.

The need for care of the child's physical well-being in school is obvious and well-recognized. Wherever, through compulsory education, the child is taken out of the home for considerable periods of time, the parents have a right to expect the best of sanitary conditions. Unfortunately perhaps, children are not made for the purpose of going to school. In the words of Dr. Terman, "We have taken the child out of its natural habitat of open air, freedom and sunshine, and for nearly one-half his waking hours we are subjecting him to an unnatural regimen; one which disturbs all the vital functions of secretion, excretion, digestion, circulation, respiration and nutrition."

With the increasing popularity of "supervised study" and other time-consuming devices of the pedagog, the daylight hours the child may spend in the open air are fast shrinking. We know that sunlight is essential for healthy growth, whether of man, animals or humans. The time is not far distant when we shall have to balance carefully the gains in educational efficiency against the possible losses in the children's health.

Since so much of the child's time must be spent in school, it is only fair to him that the building should be as sanitary and well equipped as possible. Lack of proper ventilation, overcrowding and bad lighting are perhaps not great evils in themselves, but they do lower physical vitality and give definite diseases a better chance to attack.

Ventilation is one of the most important features of a school building. Where a few pupils were grouped in a one-room school house, ventilation more or less looked after itself, but the large size of modern school buildings has gradually brought about the development of all sorts of fan and blower systems, most of which are complicated, and thrown out of kilter by opening any windows in the class rooms. But doubt has been increasing as to just how well the ventilating systems did their work.

The New York State Ventilation Commission believes

that the simplest methods are the best, either open windows or the use of various window ventilating devices with the fan system. Where a continuous inflow of out-door air from windows would interfere with the ventilating system, as is only too apt to be the case with our modern systems, all the windows in the class rooms should be opened wide between periods.

CHAPTER V.

LOOKING AFTER THE MAN ON THE JOB.

When a man is injured it is not enough to save his life. The doctor must do his best to save not only life but the ability to earn a living. If a man with a crushed hand is brought to the first-aid room or hospital, the doctor says to himself, "What can I do to make this hand fit for the man to use in his job?" instead of "What can I do to get this wound healed as quickly as possible."

The development of surgery made great strides forward after the discovery of ether and other anesthetics which gave the surgeon time to work carefully on a quiet, relaxed subject. But infection of the wounds brought this progress up with a start. Patients rallied after desperate and brilliant operations only to die of blood poisoning or gangrene. Less than sixty years ago Pasteur's discovery of the existence of bacteria and Lord Lister's application of that discovery to the operating room procedures and to the dressing of wounds redeemed surgery from the slough of disrepute into which it had fallen. So fearful was the toll of death from postoperative infection that one valuable operation was forbidden by law in France and its use discouraged elsewhere. Velpeau, the great French surgeon of the time, declared "a pin-prick is an open door to death." The records of our Civil War hospitals are full of cases of hospital gangrene, and pus was expected in the healing of any wound.

Pasteur and Lister changed all this. Sterilization of dressings and instruments, scrupulous cleanliness of surgeon, nurses and patient have made possible and safe the brilliant surgery of today. The so-called major operative surgery

has attracted most attention. Its results are spectacular, almost verging on the miraculous in many cases. The type of work is such that most doctors have been attracted to it.

But lately minor surgery has come in for its share of attention. The repair of industrial injuries is one of the most important branches of surgery of this type. Eight hundred and seventy-five thousand workers each lose four weeks or more each year from industrial accidents. Many of these accidents are preventable, but so long as they occur, doctors must do their best to make good the damage.

Most of the injuries are not serious. Thus out of 1008 workers hurt at the Pacific Mills in Lawrence during a five-year period, 2 died, 86 had broken limbs or amputations, 910 were slightly injured, and 2 suffered only from nervous shock. However, if a slight injury is not properly looked after, it may result in lasting trouble.

The first thing the surgeon learned in treating cases of industrial injury was that it paid to be the first man to do anything. Well-meant help from fellow-workmen, such as stanching of blood or taking bits of metal from the eye, too often resulted in the introduction of germs into the wound. An infected wound is much harder to heal and does far more damage to the surrounding tissues than a clean one.

Of course where proper dressings are supplied and the workers have some idea of first aid, the danger of infection is much less. But germs are no more benevolent today than they were when they thrived unknown and unmolested sixty years ago. A clean cut heals in about ten days, but if bacteria get a foot-hold in the wound, healing is slow and unsatisfactory, even though actual blood-poisoning may not occur. Even under the best of care wounds occasionally become infected.

Where a few years ago we knew little about the handling of infected wounds, the development of bacteriology has given us fairly efficient methods for their care, such as flooding the wound with Carrel-Dakin solution and the use of

dyes which injure the bacteria but not the cells of the body. Even in extensive crushing wounds, with dirt ground into them, amputation may often be avoided. The experience gained in the World War taught us that thorough flooding of the wound with Carrel-Dakin solution or other mild antiseptics, gave the body a fair chance to fight any invaders.

In order to give good service to their employees and to give the doctor a fair chance, most large plants have either a full-time physician or one on part time with a well-trained nurse to handle minor injuries in the doctor's absence. Thus it is possible for the injured man to get prompt medical attention at any time. The danger of infection is consequently slight. Instead of getting a fellow-worker to patch him up, the man goes to the dispensary.

If he were employed in one large plant near Boston and had his hand cut, say by a circular saw, a fellow-workman would cover the wound with a sterile bandage from a first-aid cabinet. When he reached the dispensary the first-aid dressing would be removed, any dead or ragged flesh cut away, the wound cleansed with ether and then swabbed with a solution of iodine, any necessary stitches taken and the wound dressed with dry gauze. In other plants the treatment would vary somewhat, but the principle remains the same—favor healing and prevent infection.

If, in spite of care or because of lack of medical attention, a wound becomes infected, all loose or dead tissue must be removed and the wound sprayed with Dakin's solution. Instead of dry gauze, gauze soaked in Dakin's solution is used for the dressing.

Broken bones are injuries sometimes met with, particularly in metal working shops and shipyards. When a broken bone heals, it does so the best it can. If the fragments are in good position, the bone is as good as new when the healing process is over. But if not, deformity of greater or less degree may be the result.

Here, again, the earlier the surgeon gets a chance to set

the broken bone the better chance he has to get a good result. He does not have the danger of infection to guard against, unless the skin has been broken, but, instead, has the danger of a bone set in a bad position or a stiff joint as a result of the long-continued quiet essential to the healing of the fracture.

In holding a broken arm motionless, he has not only to consider the positions of the two bone fragments but he must be mindful always of the risk of a stiff elbow or wrist, and set the bone not only in such a position that the break can heal, but that the nearby joint, if it becomes stiffened, will give the least possible trouble. In an injury to the joint itself, where stiffness is even more likely, this consideration becomes of paramount importance.

Sometimes it is necessary to forfeit the use of a joint in order to save the limb, as in joint tuberculosis. Here the usual position of the leg is of great importance. Should the stiff knee be in the straight position, sticking out whenever its owner sits down? Or is it better to have it bent, convenient while its owner sits down, but causing a marked limp or requiring the use of crutches when he walks? Or is a compromise between the two positions better still?

A woman sewing-machine operative, for example, would require a much greater bend at her knee than would a night watchman. All these points the surgeon must weigh for each patient and decide just which position is best.

One principle that the surgeon has learned to apply in joint work is that of over-correction. Instead of placing the bones in just the position they would normally be, he fixes them in a position a little beyond the normal. By this means any tendency of the deformity to recur would be taken care of. Where the pull of the muscles is unequal, as in the wrist, the weaker muscles should be favored. Those that lift the wrist are weaker, and consequently fractures of the lower arm or wrist are frequently fixed in the "cock-up" position, with the hand bent back, so that the

muscles pulling the wrist down not only have more work to do, but have to work at a disadvantage, thus giving the other set of muscles a fair chance.

An important factor in the avoidance of stiff joints after fractures is motion of the splinted joint from time to time, being careful, however, not to disturb the fracture itself. At first this motion should be done only by the doctor or under his directions. After some weeks it can be attempted by the patient himself. If this movement is carefully and faithfully carried out the joint will be ready for work just as soon as the broken bone is healed, instead of requiring days of limbering up. A permanent stiff joint almost never occurs with this method of treatment.

Conservation has become the watch-word of the surgeon. If he can save only two fingers on a hand, and that only by long and patient work, those two fingers must be saved if they will be useful.

There are just as many, if not more, medical hazards than surgical in industry. Certain dusty occupations favor the development of tuberculosis, in others, the dusts breathed in themselves injure the lungs. The development of new and varied chemical processes brings up new dangers constantly. The latest of these dangers is still under investigation to determine the amount of harm it may do.

In the efforts to find more efficient methods of using gasoline, chemists developed the so-called "ethyl gasoline"—gasoline with tetraethyl lead and ethylene dibromide or trichlorethylene. From the engineering point of view, the new fuel has been successful. From the point of view of health, it is perhaps not so good. Gasoline fumes alone are none too healthful, even in the open air and the addition of minute amounts of lead to them is certainly not an improvement. Whether the new fuel produces appreciably less of the deadly carbon monoxide than ordinary gasoline is an open question.

Tetraethyl lead is no new compound. In 1878 a German

investigator found that it was poisonous. Recently deaths among the workmen manufacturing tetraethyl lead called attention to the dangers present in the process and the state of New York and New Jersey forbade its use. When the excitement died down, they rescinded this action, but New York City has still kept it in force.

The problem now before the public health authorities has four aspects. Is tetraethyl lead dangerous to manufacture? Is it dangerous to handle? Is the ethyl gasoline dangerous to handle? Are the exhaust fumes from ethyl gasoline dangerous?

The first question is strikingly answered. Though only a few plants make tetraethyl lead, 10 of their employees have died from poisoning and one has become insane since June 1, 1924. There have been also a number of cases of illness. In one plant examined by the Commissioner of Labor of New Jersey, "the danger of exposure was reduced to a minimum," but a man employed in this plant died from poisoning with tetraethyl lead after the inspection had been made.

The symptoms are largely those of acute lead poisoning. First the blood-pressure and body temperature drop very low and the pulse slows greatly. Then evidences of brain injury appear, sleeplessness, staggering gait, restlessness and delusions. As the poisoning grows more severe, the patient becomes maniacal, yelling and smashing the furniture and eventually dies in complete exhaustion. In the cases where the poisoning was not severe enough to cause death, convalescence was very slow, taking from six to ten weeks.

It is probable, with more experience in manufacturing the substance, that safe methods will be developed. But from the evidence at hand, safety is far from being completely realized at present.

Aside from the danger of manufacturing tetraethyl lead is the risk entailed in mixing it with gasoline. When done

in bulk by men trained in handling the poison, there is not much danger. That is the way in which the companies handling ethyl gas aim to distribute it, so the filling-station employees will not come in direct contact with the tetra-ethyl lead.

When it comes to the handling of the ethyl gasoline, ready mixed, there seems to be no chance of harm. The tetra-ethyl lead is greatly diluted and with the pump methods of distributing gasoline there is little risk from the gasoline itself or from its fumes.

One very important question to be decided is whether the public will run any danger from this new substance in automobile exhaust fumes. Experiments and actual happenings in city streets have shown that under certain circumstances the carbon monoxide gas in exhaust fumes may reach dangerous proportions. With the increasing congestion and slowing down of traffic, both of which result in more and more production of exhaust gas, it is a pretty ticklish question as to whether we want another poisonous substance added to the air, even in minute amounts.

Lead poisoning is one of the classical examples of industrial hazards. It is commonly associated with painters, though the first recognized cases, then known as the colic of Poitou, were due to low-grade wines treated with lead. The symptoms of lead poisoning and the possibility of their association with lead had been known almost as long as the metal had been used, but this epidemic in France three hundred years ago gave the first definite evidence.

While nowadays the painter's trade is the most important of those using lead, there are many others in which the danger of poisoning is great. One of those with very serious risk is the making of storage batteries. Every automobile has one, nearly every radio fan and most of the small electric light systems. In making plates for the batteries litharge and red lead are used. The work requires little skill and therefore the labor turnover is high. These two

factors make for carelessness and ignorance on the part of the workers and indifference to proper sanitary precautions on the part of the employers.

When you look at the white shining enamel of your bathtub, it is hard to believe that red lead is one of its necessary ingredients ranging from 0.5 to 20 per cent of the total. The purpose of the lead is to give a certain degree of flexibility and prevent chipping.

Another industry in which lead is extensively used is glazing and decorating. Here very little care is given to protecting employees from lead poisoning. The last investigation by the United States Public Health Service, in 1921, showed 13.5 per cent of the workers to be suffering from the disease—just fifteen times as many as in similar trades in England. The lower rate there is due to use of a smaller amount of lead in the glaze and better protection of the workers from the fumes.

Mining lead ore and smelting it, particularly when the Scotch hearth method is used, are other important causes of lead poisoning.

In spite of these numerous other lead-using occupations, painting still takes the lead in causing plumbism. Most European countries prohibit dry sandpapering of paint, the dust from which is one of the chief sources of danger. This process is rapidly being abandoned here, not only because it is dangerous, but because it is slow.

The desire for speed has had less happy results in the adoption of the "spray-gun" for painting various articles from toys to railroad cars and for interior work on buildings. With this device the air is filled with a mist of oily drops laden with lead, and the ordinary respirator used holds back very little. The Bureau of Mines have perfected a satisfactory gas-mask, but few men would care to work eight hours a day in a gas-mask.

One of the formerly dangerous trades which now has but little risk of poisoning connected with it is printing.

In handling lead type and molten lead almost infinitesimal amounts of lead were absorbed, and when the absorption went on unchecked for years, trouble eventually developed. Most of the credit of the present healthful condition of the industry is due to the International Typographical Union.

The chief hygienic problem which faces the lead-using industries in this country is the protection of the workers from lead in the air. It has long been believed that lead which clung to the hands and was swallowed was the chief source of plumbism, and that washing before meals was practically the only safeguard necessary. But this does not prevent lead poisoning.

The departments of physiology and industrial hygiene at the Harvard Medical School have been at work for several years on lead poisoning in all its aspects, and have succeeded in working out a very satisfactory treatment, the giving of phosphoric acid by mouth. There are several types of lead poisoning, some acute, others very chronic. The type depends very largely on the susceptibility of the individual, just as some people can eat cucumbers and milk together without any trouble.

There may be nothing more than weakness, general ill-feeling, vague aches and pains, dizziness and headache. Loss of appetite is very common. Often nothing may be noticed for months beyond a metallic taste in the mouth and a vague sensation of nausea.

The best known symptom is "painter's colic," intense colicky pains usually in the vicinity of the navel, which come on suddenly and last for varying periods. Perhaps the most useful single sign in making a diagnosis of lead poisoning is the lead line along the gums. This is a row of fine blackish-blue dots in the gums just beyond the line of the teeth. It is caused by deposits of lead in the cells of the deeper layers of the gums, where sulphides from decomposing particles of food about the teeth change the metallic lead to black lead sulphide. Only one other substance—bismuth, which

is very rarely met with—gives this line. Unfortunately not all cases of lead poisoning show it. Its presence proves a lot; its absence proves nothing.

A blood smear may show a characteristic change in the red blood cells called stippling, in which some of the cells are filled with bluish granules. Again the absence of this sign is of no value. Chemical tests for the presence of lead in the urine are a great help in diagnosis.

Chronic lead poisoning, due to the gradual absorption of lead, usually attacks either the digestive system or the nervous system, with a certain amount of anemia, increased blood-pressure and increased size of the heart. The symptoms of colic, nausea and constipation may be due in the last analysis to changes in the nerves of the stomach and intestines, so that lead is probably chiefly a nerve poison.

When it does attack the nervous system so as to cause symptoms directly referable to it, about the first thing noticed is a dull headache over the eyes. Then patches of neuritis appear, joint and muscle pains a good deal like rheumatism, and may go on to difficulty in controlling the finer movements, such as those of the hands and fingers. Frequently, if exposure to lead is not stopped, definite palsies develop. The commonest of these is wrist-drop, due to inability of the muscles of the forearm to straighten out the hand or the wrist.

Chronic lead poisoning usually comes from very minute amounts of lead acting over a long period of time. As a general rule, the lead has been absorbed by way of the digestive tract. This is the least dangerous way to take in lead, as a good deal may pass through unabsorbed, and the liver removes from the blood and excretes in the bile a large part of that which has been taken up, so only very small amounts get into the general circulation.

Where lead is breathed in as dust or fumes, however, a much larger amount is absorbed, and there is no buffer like the liver interposed between the lungs and the general

circulation, so what lead gets in is free to attack any point in the body. If anyone at all susceptible to lead is exposed to the dust or fumes for any length of time, acute rather than chronic lead poisoning is apt to develop.

This acute form attacks the brain very often, with loss of control over the muscles, delirium, and convulsions. In the severest cases it may progress to blindness, paralysis or death.

Another of the important problems, so far as the severe industrial accidents are concerned, is that of resuscitation. In our larger cities, hardly a week goes by without a number of severe cases of gas poisoning or electric shock, with not a few drowning accidents to swell the total of sudden death from respiratory failure. In electric shock or drowning the doctor faces a very different problem from the wasting diseases he usually has to fight. Instead of a broken down body with lungs, heart or kidneys gone beyond repair, he usually deals with a perfectly healthy man whose breathing has been stopped by immersion in water or by electric shock. The problem is to start breathing again, and once this has been done all is well, provided there have been no unusual complications.

In the words of Dr. Henderson of Yale, the greatest authority on resuscitation, it is like cranking an automobile when the engine has stalled and the self-starter is out of order. The essentials of artificial respiration are immediate application and tireless endurance. Even if no pulse can be detected and the person is to all appearances dead, artificial respiration should be kept up for three to four hours, unless natural breathing has begun or the stiffness of death set in before that time. Not a moment should be lost in beginning the treatment; a minute or two at the start may mean the difference between life and death.

After a vast deal of experimental work and actual use, the Schäfer prone pressure method of artificial respiration has been found the best. Its advantages are four-fold. There

is no apparatus to wait for, it can be applied by one person without assistance, it is the easiest and least tiring of the various methods, and it most nearly imitates natural breathing.

Where men are working in small groups and often far from a hospital, as is often the case with electric linemen, any method on which life depends must be simple and ready for instantaneous use.

The American Medical Association has appointed commissions to study the problems of resuscitation and the results of their studies have been of very great value to the electrical industries. Invariably the prone pressure method has been found the most suitable.

Once the victim has been removed from the current if shocked, the water if drowned, or the gas-filled room if overcome by gas, one line of procedure fits all three cases. Place him lying flat on his stomach, with one arm stretched out full length; the other arm outstretched but bent at the elbow, with the face resting on the forearm. If the jaws can easily be opened, make sure the mouth is clear; if they are tightly closed, do not waste time trying to open them. The nose will serve for air passage.

The rescuer now kneels, facing the head straddling the victim's back, with knees just below his hip bones. He then places the palms of his hands on the small of the back with the fingers resting on the ribs. The tips of the fingers should be just out of sight on the sides, and the little fingers over the last ribs.

When the hands are in position, the arms are kept straight as the rescuer throws his weight forward. This motion should not be sudden and ought to take two to three seconds. Otherwise some of the internal organs may be injured. The lungs are now ready to suck in air when the chest can expand again. At the end of this forward swing, the resuscitator immediately swings back, completely removing the pressure. The chest wall is very elastic and expands as soon as the

compressing force is removed, and the diaphragm drops back toward the abdominal cavity. The resulting expansion of the lungs causes air to be sucked in. This is as close an imitation of the natural breathing as can be obtained. The double movement of compression and release should be repeated twelve times a minute.

Lung motors and pulmotors, instead of making the lungs suck in air, force air in. Their only advantage is that they save some hard work; in the hands of inexperienced people they are worth very little.

If death from electric shock were invariably due to stoppage of breathing there would be little excuse for its causing death. However, it may affect the heart as well. There are several facts about electric currents that must be kept in mind. If the parts of the body making the contact are wet the shock will be much more severe than if they had been dry. This is the reason for the frequency of severe or fatal shocks from the ordinary low voltage light circuit in bath-rooms. If the current passes from foot to foot, little harm results other than the burns at the points of contact. But if the current goes from arm to arm, or arm to leg, so that it passes through the chest, shock will occur.

A low voltage shock usually does not affect breathing, but it so stimulates the heart that the normal beating is replaced by a rapid quivering of the heart muscle, which is utterly ineffectual in pumping the blood and maintaining the circulation. After a very few moments death results. Currents of 220 volts or below have this effect on the heart, while those of more than 1000 volts have but little action on the heart, disorganizing the breathing mechanism instead. It is to this latter class of cases that artificial respiration is applicable. Yet it ought to be used in every case, for the great majority of shocks are due to high-voltage currents—just where the method works best—and even with low voltage the breathing may occasionally be stopped instead of the heart.

The value of artificial respiration in drowning is too well known to need mention. The prone pressure method is the most practical to use here as well. It is not at all uncommon for natural breathing to stop once it has been reëstablished. When this happens artificial respiration should be resorted to again without delay.

A further refinement in artificial respiration, not available except in the best equipped of hospitals, though it should be more commonly used, is substitution of a mixture of carbon dioxide and oxygen for air. The important thing is to supply oxygen to the body; the lack of it is what does the harm when breathing ceases. Now the air is one-fifth oxygen. The rest is of practically no value to us. Unfortunately neither pure oxygen or air enriched with oxygen is a stimulant to breathing. It may be just the opposite. Anyone who has much experience with fires knows that firemen who have been working in smoke or fumes often collapse when they come out into the fresh air. The same thing may happen to members of mine rescue crews.

The stimulus to breathing that Nature has provided for us is carbon dioxide, the gas given off by the lungs under normal circumstances. Now when short of oxygen, whether because of gas or fumes in the air or because of great exertion, we breathe deeply and rapidly, giving off large amounts of carbon dioxide with every breath. It is flushed out of the body. Then when the oxygen lack is made up, whether by fresh air or oxygen, the result is marked depression of breathing and not enough carbon dioxide is present to start things up again.

If we want to get in the largest amount of oxygen in the shortest possible time and also stimulate normal breathing, the logical thing to use is a mixture of oxygen and carbon dioxide. This combination is particularly valuable for use in cases of carbon monoxide poisoning, whether from illuminating gas, coal gas, automobile exhaust fumes or mine explosions.

Even if the victim of carbon-monoxide poisoning is brought out into the open air before he becomes unconscious, the gas is eliminated from the blood very slowly and a day or two of splitting headache and intense nausea are in store for him. However, if he is given a mixture of carbon dioxide and oxygen by feeding the gases into a mask over his face, he is usually back on the job in an hour or two without any ill-effects. The deep breathing and plentiful supply of oxygen rapidly get rid of the poisonous gas. In severe cases, where death seems imminent, the prone pressure method of artificial respiration combined with inhaling the oxygen-carbon dioxide mixture works apparent miracles.

The same mixture has been used very successfully to shorten the period of recovery from ether and to revive persons unable to stand the shock of anesthesia and operation.

With full use of the methods at our disposal, the number of deaths from electric shock, drowning and gas poisoning can be cut down considerably, just as care of minor injuries has reduced the number of severe infections with their disastrous results.

CHAPTER VI.

SLEEP AND SLEEPLESSNESS.

Sleep is one of the most necessary things in life. When sleep comes to us readily, as it usually does, we think very little of it, but once let insomnia fasten on one, sleep becomes the most necessary and most desirable thing in the world.

The very commonness of sleep, the great proportion of our time normally spent sleeping, show that sleep is a physiological process of great importance. When we have been doing bodily or mental work any length of time, the increasing weariness causes a desire for sleep that, even though put off a while by drugs such as strong tea or coffee, in the end becomes overpowering. The stories of soldiers going to sleep on the march are by no means myths. We can fight against this desire for sleep, but it is a losing battle, as anyone who once starts to nod knows.

One of the most refined of the Chinese methods of torture is the prevention of sleep. The victim lives only five to seven days. Yet we can live nearly as long without water and five or six times as long without food.

What is sleep? Loss of consciousness and voluntary motion, you say. But that is only describing the two most obvious things about sleep. Even if we go still further and say that the muscles are relaxed, the pulse and respiration rates lowered, reaction to outside sounds and sensations absent or lessened, we have not gotten very far.

We know that when the blood supply to the brain is lowered to a certain degree, unconsciousness results. Dr. Mosso, an Italian physiologist, made a number of investigations on men who had had portions of their skull trephined out for various reasons, and so had only a soft scar over part of

their brain. By the amount of pulsation of this scar and the pressure against it from within, the amount of blood circulating through the brain could be determined, at least relatively. Mosso found that there was always less blood circulating through the brain during sleep or unconsciousness than during consciousness.

The blood that does not go to the brain during sleep has to go somewhere else. An ingenious device called the plethysmograph, which simply is an air-tight case fitted about a limb and connected with a tambour and lever, can be used to determine changes in volume of the part encased in it. It is fairly safe to say that any rapid change in volume is due to a change in the amount of blood circulating through the part. With this instrument Mosso found that more blood circulated through the extremities during sleep. A shake or touch just sufficient to cause awakening is followed by lessened flow of blood in the arm and a rise of general blood-pressure, which means more blood for the brain; and in awakening the sleeper the same changes are carried still further.

So sleep is accompanied by or caused by a lessened flow of blood to the brain. But even here we have no real explanation.

Throughout the animal kingdom we find a more or less constant rhythmical activity, an alternation between exercise and rest. The vast majority of birds and animals go to sleep as the dark comes on, and those that are night prowlers, such as the owl and alley cat, usually sleep by day. Just so most of us sleep by night, but a few of us who have to work at night and sleep by day reverse the normal rhythm.

Even in our own bodies the heart beats and then rests, so for all its steady pumping it actually works only about a nine-hour day in the average person. A muscle contracts, with the production of acid fatigue products, and then rests, while the lactic acid formed by the contraction is changed back to its former state.

In this rhythmicity we probably have the secret of sleep. Through the day we are active and fatigue products accumulate, accompanied by a fall in what might be called potential energy—or more simply, loss of "pep." When the store of potential energy nears exhaustion, that is, when we feel tired, we tend to go to sleep. During sleep the fatigue products are taken care of and we wake refreshed, ready for a new day.

In some ways the process is a good deal like that of a voluntary contraction of muscle. The muscle when it begins to get tired makes smaller contractions until it lies motionless in spite of the orders sent along its nerve, from the brain. But if, when the muscle lies apparently useless, it is directly stimulated, say by an electric shock, it contracts nearly as well as ever for a while. In other words, the nerve has become unable to stimulate the muscle to contract once a certain stage of exhaustion has been reached, even though the muscle has considerable reserve strength. Nature steps in and prevents extreme exhaustion.

Sleepiness comes on long before actual exhaustion of the brain, as any one who has worked hard and late by sheer will power or the aid of black coffee well knows. Though the reserve energy varies in different people, it is there to some extent in all. However, it is kept on hand by Nature for emergency use, and not to be steadily drawn against.

Fatigue and sleepiness are the signals that Nature provides to warn us when we begin to draw on this reserve of nervous energy. If we habitually neglect this warning, exhaustion of the highest function of the brain follows—that is, the inhibitory function, the same one dulled by alcohol—and the unchecked associative memory keeps up a constant flow of ideas through the consciousness, which in turn causes mental excitement and sleeplessness. Thus a vicious circle is established, and we find that all too common condition, where the people most in need of sleep are too nervous and excited to get it.

This state is typical of insomnia, and the constant lack of nervous energy is the cause of the irritability and bankruptcy of thought that characterize that state.

The value of the various methods of inducing sleep, such as counting sheep and kindred devices, is that the flow of ideas which the tired brain cannot readily head off, is checked, and the break gives a chance for sleep to come.

Everyone has had the experience of awaking tired after sleep troubled by dreams. Dreams actually use up nervous energy and sometimes muscular as well. Even animals dream. My dog has been lying quietly at my feet. Suddenly he sniffs the air, quivers and thrashes his legs about as if he were hunting in his sleep.

Insomnia has many causes, such as pain, fever, or irritating cough, but the most important single cause is worry. Insomnia of the worry type is an important factor in the so-called nervous breakdown so often blamed on overwork. Overwork may have caused a few nervous breakdowns, but worry and mental turmoil have far more to their credit. When the worry prevents sleep, or spoils what sleep can be obtained with dreams that revive or foreshadow the troubles of the day, it is not long before the reserve nervous energy dwindles to nothing. Then we find the vicious circle in operation—the exhausted brain unable to protect itself from the flow of thoughts that prevent the needed rest.

There is no medicine for insomnia. True, there are various drugs that cause sleep, but they work no permanent cure. The thing to do is to strike at the source and restore the habit of normal sleep. The cause of worrying can often be removed, or can be made rather insignificant provided it is looked at dispassionately. Anything that is monotonous and breaks the flow of ideas is of value.

Often by simply lying quiet and not worrying about the sleeplessness, keeping the mind vacant if possible, sleep can be coaxed where it cannot be driven.

CHAPTER VII.

THE WAY GERMS WORK.

THE doctor is the only man I know of who does his best to spoil his business. Not only is he busy in looking after those who are sick, but in trying to keep healthy those of us who are well. One of the biggest problems to be solved—in fact, the one to which eventually each separate disease problem leads—is why we get sick at all.

The causes of many diseases are known: such as the typhoid bacillus for typhoid fever, the tetanus bacillus for lockjaw, or the plasmodium for malaria. But the mechanism by which the typhoid bacillus produces typhoid fever and not malaria, or why some people have more severe cases of the disease than others, is little more than guessed at. In such diseases as cancer or high blood-pressure, where the cause is as yet unknown, we are even more at a loss.

In the first place, there are many thousands of bacteria and simple forms of animal life. Only a few of these are capable of producing disease. Many germs are helpful, such as nitrogen-fixing bacteria in the soil, the yeasts familiar to makers of bread and brew, or the germs that give their distinctive flavors to Camembert, Limburger and other cheeses. Others are always with us, but do no harm. One bacillus normally lives between eye and eyelids, and we would be none the wiser were it not for the bacteriologist and his microscope. The mouth and nose are inhabited by numerous bacteria. Water, fresh or salt, and soil are alive with them. Why is it that certain ones can cause any degree of trouble, from slight illness to death, while others are harmless or even necessary?

The bacteria can be divided into three great classes—the ones which cannot under any circumstances grow and multiply in the body of a given animal and therefore are harmless; the ones which can develop in animals only under specially favorable conditions and the ones with marked ability to invade the body. Most of the disease-producing germs are in the second class, needing suitable conditions for their development.

The various factors which determine the ability of a germ to invade the body are summed up in the term "virulence." Our bodies are a battlefield, with the forces of bacterial virulence on one side and bodily resistance on the other.

The resistance of the body depends greatly on the path by which the bacterial invasion occurs. The unbroken skin is a harder nut to crack than the Hindenburg line and while cuts and breaks give germs a foot-hold, as a rule they are soon driven out by the reserve defences of the body, the white blood cells. But if dead, crushed tissue is present, as in many of the war wounds and in street accidents, the germs which get into it have an excellent base in which to multiply undisturbed and from which to attack the living tissues.

The stomach with its highly acid digestive juices forms a barrier for many of the bacteria which are swallowed, but once beyond that danger point, the twenty-odd feet of delicately lined intestine provide a happy hunting ground for such germs as those of typhoid fever, dysentery or cholera.

A defense that most of us would probably not stop to consider is that provided by our body temperature. Yet this is really of importance. Germs do not wear clothes nor live in heated houses, so temperature makes a good deal of difference to them. Take for example the difference between our body temperature and that of birds—only a few degrees—yet the bacillus that produces tuberculosis in birds is harmless to man and *vice versa*.

In most diseased conditions we have an increase over the

normal body temperature; we are feverish. This fever is one of the attempts at self-defense made by the body. The various chemical reactions involved in fighting the invading germs are speeded up by the greater heat, and the germs may find things too hot for them in both senses of the word. Thus a fever is not an unmitigated evil, to be lowered at any cost.

Fevers end differently, in some there is a sudden drop of temperature to normal. This so-called "crisis" may take place in a very few hours. On the other hand, the temperature may come down slowly and perhaps linger just above normal for a few days. An ending of this type is called "lysis." Those diseases ending by "crisis," such as pneumonia, are usually ones in which very little damage has been done to the tissues by the germs, and when immunity has been established the trouble is over. In cases ending by "lysis," on the other hand, usually extensive damage has been done, so when the germs have been defeated, the injured tissues have to be repaired.

Just as important as the point of attack is the number of the invading germs. A few varieties such as anthrax or plague can cause disease if only a very few enter the body. As a rule, a fairly large number is required to make trouble, and the larger the number is, the surer trouble is to come.

By what mechanism can a very few anthrax germs kill a healthy man, when hundreds of typhoid germs might only make him sick? The two necessary things for a successful disease germ are not so different from those of a good business man—persistence, that enables it to stay on the job and not be overcome by the body defenses, and punch, that puts the body on the defensive and keeps it there.

The anthrax germ, luckily a rare one around here, is one of the Henry Fords of the bacterial world. By analyzing its means of defense and offence we can get an idea of what makes a successful germ. In the first place, it has a mechan-

ism by which it can live for long periods under unfavorable conditions. Just as a woodchuck hibernates, the anthrax germ forms a spore. Then when the surroundings are once more favorable, the spore changes back to the bacillus. The spore may live for years in soil. It stands fairly strong solutions of carbolic acid. It requires boiling water to kill it, whereas most germs can be killed by temperatures of 135° F.

Once in the body the anthrax germ forms a shell about itself which protects it from the attacks of the body's white blood cells and fluids. Thus it can grow relatively undisturbed in spite of all efforts of the body to defend itself. It multiplies rapidly, so that even a day or so after the first infection the body is swarming with them. It can grow in the circulating blood, by which it is swept to all parts of the body, from liver to brain. It produces poisonous substances which depress the bodily activities.

However, the virulence of the bacteria is not the only point that decides our fate. As a rule the resistance of the body is a fair match for the invaders. Moreover, few germs are as well-equipped trouble-makers as the anthrax bacillus.

Our first lines of defense—the skin, the acid secretion of the stomach and the body temperature—are usually enough. When they break down, the white cells of the blood do their best to surround and destroy the invading germs. Meanwhile the body cells are stimulated by the presence of the bacteria and the poisons they secrete to form substances known as antibodies. As to what antibodies are, we know little. But what they do is another story. One type of antibody neutralizes the poisons secreted by the bacteria. Another type precipitates and renders harmless any of the bacterial protein which may have been set free. Still another dissolves the germs. As a rule, not all those antibodies are produced against any one germ, but only those best suited to resist its attack.

The particular symptoms we get in any given disease are

the result of the interaction of the attacking bacteria and the body's defensive forces.

Each variety of disease-producing germs possesses certain peculiar characteristics, which determine not only its mode of attack, but the result of its attack. Thus the diphtheria bacillus is adapted to living in warm, moist, dead tissue. It finds suitable conditions in the throat. When it lives on material containing protein, it secretes a powerful toxin. The symptoms of diphtheria are due to the action of the toxin on the cells of the body. The body tries to protect itself by forming antitoxin. Now we know enough to help out the body by injecting into it ready-made antitoxin. Indeed, we can go a step farther, and through the Schick test find out those persons susceptible to diphtheria and then enable them to become resistant to the diphtheria bacilli. This last is done by injecting the toxin-antitoxin mixture, which causes the body to form antitoxin and so have it ready for immediate use should the germs attempt to gain a foot-hold. Perhaps some day we will be able to help as efficiently in all diseases.

To come back to our original question, the ability of a germ to cause disease in a given person, the severity of the symptoms, and the outcome all depend on the balance between the virulence of the attacking germ and the protective mechanism of the body.

PART II.

IMPORTANT DISEASES.

CHAPTER VIII.

BLOOD POISONING.

A BROKEN blister—blood poisoning—death.

Such a tragic sequence is fortunately rare, though far more frequent than it should be. Why is it that a hardly noticed break in the skin may lead to sudden death? And more important still, how can we prevent serious results from these apparently slight injuries?

The skin itself is one of the strongest defences of the body. Think how many diseases enter by way of the nose or mouth; how few by the unbroken skin. Pneumonia, whooping-cough, typhoid fever, consumption, almost all the common diseases must attack us through the mucous membranes of the nose, mouth, or intestinal tract, as they are unable to penetrate the skin. But if a break occurs, this barrier loses much of its value. For germs to get in, only a very small opening is required.

Once through the skin, one of several things may happen to the invading bacteria. They may be immediately disposed of by the white blood cells; in this case the wound heals normally. They may resist the white blood cells a short while; here we have the usual infected small wound—redness, pain, and perhaps a small amount of pus, followed

by healing. They may overcome the local defences and invade the tissues, giving the ordinary picture of blood poisoning, so-called.

This invasion by the germs takes place along the lymph channels which drain the tissue juices back into the great veins. It may be stopped at the lymph nodes, masses of white blood cells acting as filters for the lymph, or it may work past them, leaving the germs free to enter the blood.

The outcome of the invasion depends on the relative powers of the body to defend itself and of the bacteria to attack, just as a thin board can stop a sling-shot, but sheet iron is needed to stop a rifle ball. The size or character of the wound has not much to do with the question. In one case, a broken blister leads to death, in another, a foot-long gash from a broken windshield heals without trouble. Sometimes the organism that gets in is of such virulence that no one can withstand it. A number of years ago an athlete in the pink of condition was competing in the Caledonian games. He ran against a hatpin and in forty-eight hours he was dead. In other words, though his body defences were in first-class condition, they were powerless against the very virulent germs that got in through the scratch.

If we follow through a typical case of blood poisoning, it may help to make things clear. A man is walking along and scratches his finger on a barbed-wire fence. At night the scratch is somewhat painful, looks red, and throbs a little. He does not sleep well. In the morning he feels mean and has a fever. The edges of the scratch pout and there is a little thin pus present. The finger is swollen, throbbing and very painful. Little sore lumps are present in the crook of his elbow and in his arm-pit. Through the day streaks appear along his arm. The swollen, hard lymph nodes at elbow and arm-pit become very tender. Gradually the whole arm swells and reddens, the fever grows worse, and delirium may set in.

When the doctor is called, he will make long slashes in

the hand and arm and put in drains. Thin bloody pus may drip out and the pain will be much relieved. If drainage has been established early and the germ is not too virulent, recovery gradually occurs.

This is what we commonly call blood poisoning. The germs do not get beyond the lymph nodes, which act as filters for the lymph coming from the tissues, but their poisonous products do, causing fever and other constitutional disturbances. Technically this case I have just described would be diagnosed lymphangitis with toxemia—inflammation of the tissue spaces and lymphatics with poison in the blood.

The ending "emia," means "in the blood." Toxemia is poison in the blood; common with scarlet fever and diphtheria. Bacteriemia means germs in the blood, but doing most of their harm somewhere else. There is a bacteriemia in typhoid fever. Septicemia means bacteria growing in the blood and forming poison there. Pyemia is masses of pus-producing organisms in the blood. One of these last two, septicemia or pyemia, is the cause of death in most fatal cases of blood poisoning.

Which it will be, depends on the kind of germ that is making trouble. Streptococci, which look like a string of beads under the microscope, do not form much pus and produce septicemia, death occurring from the poisons formed. Staphylococci, arranged like a bunch of billiard balls, and about the same size as the streptococci, cause pyemia, forming abscesses throughout the body, particularly in the marrow of the bones. Here the harm is caused not only by poison from the germs, but also by the destruction of tissue by the abscesses.

The staphylococcus received a good deal of notoriety several years ago, for it was the germ which killed Calvin Coolidge, Jr. It usually causes only local infections, such as boils and carbuncles, but occasionally a particularly virulent one breaks through the wall of white blood cells

with which the body tries to hem it in and gets into the blood stream. Wherever a mass of germs is swept by the blood an abscess forms. Very soon after the President's son became ill an abscess was found in the marrow of one of the bones in his left leg. The germs were obviously carried here by the blood, since the infected blister which caused the trouble was on the right heel. It was to drain this abscess of the bone and so relieve the intense pain and absorption of poison that the operation was performed. But the germs had done so much damage and had been so thoroughly distributed through the body that relief of this one focus counted for little.

It is fairly easy to tell whether the bacteria have broken past the last line of defence of the body, the lymph nodes, and entered the blood. A small amount of blood is taken with great care to ensure cleanliness and sterility from a vein in the arm and added to a flask of special broth. This flask is incubated and examined at intervals for the presence of bacteria.

There is one form of septicemia which is very chronic, lasting sometimes for months, but practically always fatal. It is caused by a streptococcus closely related to the common one, but much slower in action. These germs generally get in by way of infected tonsils and grow on the valves of the heart. Little masses of the growing germs are broken off from the valves and carried by the blood to different parts of the body, causing local injury. The outstanding injury is that to the heart, and death is as a rule due to heart disease. This form, however, is entirely distinct from ordinary blood poisoning.

We have seen how serious a condition blood poisoning is. The ideal treatment of blood poisoning, as of all infectious diseases, is to destroy the cause of the trouble promptly. Then the body can repair the damage. In a few diseases only can we do this. Thus quinine is a sure cure for malaria, killing off all the malarial parasites in the body. In a few

other diseases we can do it, but the risk to the patient is too great. There is a chemical substance called optochin which quickly kills off the pneumococcus—the germ causing pneumonia—but this drug may do more harm than the disease itself, causing blindness or death.

The aim of research workers on this subject has been to find substances which will do the maximum amount of harm to the invading germs with the least amount of harm to the person invaded.

In the search for satisfactory weapons against disease the dyes have come in for their share of attention. Dyes and chemically related substances have been used indirectly and directly. In the indirect use, various antiseptic substances are combined chemically with the dye. Examples of this are salvarsan or "606," a compound containing arsenic and of great value in treating syphilis; and the antiseptic, mercurochrome, where there is mercury combined with a dye.

The use of the dyes themselves is just beginning. The one we know most about is gentian violet. This violet stain has long been used in combination with iodine by bacteriologists to distinguish between two great groups of germs. It is called the Gram stain after its inventor. Some germs stained in this way remain violet in spite of washing with alcohol, others lose the dye. The first are called Gram-positive, the others Gram-negative. The important Gram-positive organisms are the pus-producing and poison-producing germs, the streptococcus of blood poisoning among them, while the Gram-negative ones are those that cause such diseases as typhoid fever, dysentery and infections of the kidney.

Dr. Churchman of Cornell believed that this difference in behavior of the germs toward gentian violet might go further than their mere staining properties. He mixed different dilutions of the dye with the substances on which bacteria are commonly grown, planted the germs on them and then

watched for results. When the experiment was over, he found that the Gram-negative germs—the common colon bacillus, which lives in the intestines of all of us, was the one he used—were not much affected by the presence of the dye unless its amount was relatively large. On the other hand, the Gram-positive bacillus of anthrax was prevented from growing when there was only 1 part of dye in 1,000,000 parts of the medium.

You will notice I said "was prevented from growing," instead of "killed." Technically this result is called bacteriostasis. A substance that prevents germs from multiplying is called bacteriostatic, one that actually kills them is called bactericidal.

In stronger concentration the gentian violet actually kills the germs. However, the bacteriostatic action of the dye is generally all that is needed in the body, as if the invading bacteria are prevented from multiplying the ordinary defences of the body, that is, the white blood cells and certain chemical elements of the body fluids, can take care of them. One of the great advantages of gentian violet is that it is entirely harmless to the cells of the body in the strengths usually employed.

The common germs can be arranged in groups more or less accurately according to their behavior toward gentian violet. The common germ of boils and abscesses, the staphylococcus, and the germs of anthrax and other related forms, are very susceptible. The streptococcus, very often in infected wounds and the commonest cause of blood-poisoning, is somewhat susceptible. The Gram-negative bacilli—the germs of typhoid, dysentery, or the colon bacillus normally present in our intestines—are affected only by relatively strong solutions of the dye.

These last, however, are attacked by other dyes which stain Gram-negative organisms readily. Acid fuchsin and acriflavine are the ones commonly used.

Much as it has helped us, dye therapy has its limitations.

Where a cure is perhaps most needed, in blood poisoning, it does not work. Dr. Churchman was careful to warn against expecting too much, or indeed anything, for the use of gentian violet in blood poisoning, as the dye is rapidly destroyed by the blood. In spite of this warning, gentian violet has been injected into a vein as a last resort in a few cases of blood poisoning, apparently without doing much good though a few favorable results have been reported.

Mercurochrome, another antiseptic made of a combination between mercury and a dye, has been injected in a number of cases of blood poisoning, though here again it is hard to tell whether the results warrant its use. In fact, outside of rest, plenty of fluid internally and prompt drainage of any pus which may form, there is little that can be done for the sufferer.

It is far better to avoid blood poisoning than to try to cure it. The first and most important rule to follow is—be clean. A clean wound rarely becomes infected. The next rule is—keep any cut or blister clean. It is a good plan to let a cut bleed a little, so that the blood will wash out any germs that have gotten in. Then wash thoroughly around the wound and remove any dirt or foreign material in it. If you get a blister, prick it with a needle which has been held in the flame of a match and let out the water. Then, if the blister is where it is exposed and apt to get dirty, cover it with a little clean gauze or freshly-washed old handkerchief and then stick across a strip of adhesive plaster.

A cut can be covered in the same way, or with a bandage, preferably a sterile one. It is never wise to apply adhesive plaster directly to a break in the skin.

Personally, I use iodine on all cuts, scratches or pimples. It may not be necessary, but it does little harm, and I would rather use it ninety-nine times where it is not needed than have blood poisoning develop once because it was not used. I suppose any other disinfectant would do as well, but iodine is sure, rarely does harm, and the stain shows just where it

has been applied. A few people with very sensitive skins have a good deal of trouble from iodine. For them some other and more mild disinfectant is advisable. A 2 per cent aqueous solution of mercurochrome is preferred by many. Of course the antiseptic may not penetrate far enough to kill all the germs, and so will not invariably prevent trouble, but I would sooner take my chances on a cut cleaned and treated with iodine or mercurochrome than one simply washed clean.

Above all, do not lose hope when dealing with a case of blood poisoning. The disease is very serious, but prompt and energetic surgical treatment gives more than a fighting chance, provided the germs have not already broken past the lymph nodes and into the blood stream. Even then recoveries have occurred, though very, very few.

CHAPTER IX.

SCARLET FEVER.

THERE are few of the quarantine placards for which we have more respect than the red one warning of a case of scarlet fever, and with good reason. Not only the disease itself, but the long train of disorders to which it may give rise, makes it one of the most dangerous of the so-called "children's diseases." Since 1922 Massachusetts has had 44,897 cases of scarlet fever, with 579 deaths, and these were not particularly bad years. The disease is chiefly prevalent in the fall and winter months and as the warm weather comes on both the number of cases and the severity of the individual attacks tend to decrease.

Perhaps the commonest complication in scarlet fever is running ears, and the inflammation thus set up is the outstanding cause of deafness and deaf-mutism in children previously normal. The kidneys are other organs upon which the disease gets in deadly work, frequently causing Bright's disease with death or permanent invalidism. The joints are often attacked, with resultant rheumatism. Many cases of chronic heart trouble date back to an attack of scarlet fever.

It is natural with so severe a disease that attempts to discover its cause and a cure were begun many years ago and have been kept up ever since. Many theories as to the cause have been advanced, only to be discredited when careful studies were made.

One fact stood out, however. In nearly all the complications of scarlet fever, whether running ears, severe anemia, or heart trouble, a germ called Streptococcus hemolyticus was found to be present. That name is simply the Greek

way of saying "little spheres arranged in chains which can destroy the red coloring matter of blood." There are many varieties of this Streptococcus hemolyticus, but all of them have a bad reputation. Some are the ones that usually cause blood poisoning, some produce septic sore throat, others cause erysipelas. They attack animals as well as man.

But the varieties found in scarlet fever and its complications (though even here there are slight differences) are pretty much a class by themselves. However, definite proof that Streptococcus hemolyticus caused scarlet fever has been lacking until recently.

In 1923 Dr. Dick of Chicago and his wife, also a physician, gave the world good evidence that a certain kind of Streptococcus hemolyticus caused scarlet fever. Scientists are a pretty skeptical lot, and the Drs. Dick knew that they would have to present a good case against the streptococcus before it would be found guilty of producing scarlet fever along with its numerous other misdemeanors.

Robert Koch, the discoverer of the tubercle bacillus, and who did more than any man except Pasteur to develop the science of bacteriology, laid down three conditions which had to be fulfilled before any given organism could be convicted of causing a given disease. These so-called "Koch postulates" are simple and yet highly exacting. They are: that the germ must be constantly or nearly constantly present in the diseased tissues; it must be isolated and studied in pure culture, free from all other germs; and it must be able to produce the disease under experimental conditions.

The first postulate was easily satisfied by the Dicks, as the streptococcus was commonly known to be present in scarlet fever. The second also was satisfied fairly readily. The last offered more difficulty, but several nurses who had never had scarlet fever volunteered to have swabs loaded with the suspected streptococci rubbed on their tonsils and subsequently developed typical scarlet fever.

One hitch in their chain of evidence was that two types of the streptococcus, one of which was able to ferment a sugar-like substance called mannite, and one which could not, were found in scarlet fever cases. But it was found that both could cause the disease under experimental conditions.

In the course of their experiments, the Drs. Dick had to rule out the existence of some other very minute virus which might be carried by the streptococcus and be the real cause of the disease. In order to do this, they grew the streptococcus in a fluid, and then filtered this fluid through a filter so fine that the bacteria were held back. From what we know of disease-causing viruses smaller than the ordinary run of germs, they can go through a filter of this type. So if any were present and responsible for the disease, the filtrate should work just as well as the streptococci in causing the disease. But this was not the case. The streptococci could cause scarlet fever, the filtrate could not.

But though this filtrate could not cause scarlet fever, it did contain poisonous substances formed by the streptococci. When this filtrate containing the toxin was diluted down 1000 times, 1 drop injected into—not beneath—the skin sometimes caused a zone of redness about the site of inoculation and sometimes did not. Not very exciting, apparently. But on going over the past history of the people who had been injected, the Drs. Dick found that none of those who had had a typical attack of scarlet fever showed any redness. Practically all the patients getting well from scarlet fever showed no redness after an injection. Evidently there was something present in the bodies of these people which protected them from any irritant action of the toxin.

Now scarlet fever is a disease which we do not ordinarily have twice. One attack as a rule gives immunity which lasts throughout a person's life. So we find that these people immune to scarlet fever because of a previous attack give no reaction to the Dick test, as the injection into the skin of a minute amount of his toxic filtrate is called.

Of the people who had never had scarlet fever, a little under one-half gave a negative test also. But long and careful studies of the contagiousness of scarlet fever have shown that less than one-half of the children and a still smaller proportion of adults exposed to the disease catch it. Here again an apparent discrepancy is explained for us.

Not all persons who have a positive reaction to the Dick test show it in the same degree. The test is done in much the same way as the better known Schick test for immunity to diphtheria. One drop of the diluted filtered toxin is injected into, not under, the skin of the arm with a small syringe and hypodermic needle. A drop of salt solution is also injected as a control. If the test is negative, and the person immune, nothing happens. After four to six hours, if the test is positive, redness and sometimes swelling of the skin appear, reaching their maximum in eighteen to thirty-six hours, and then fading out after forty-eight hours. A slightly yellowish area, sometimes scaling a little, remains for a week or ten days.

The degree of severity of the reaction apparently indicates roughly the susceptibility of the person tested. That is, a faint red blush about the point of inoculation shows only a slight degree of susceptibility, while a person in whom the injection caused a reddening of the skin a couple of inches across, with a little swelling in the center, would in all probability catch scarlet fever after only a slight exposure.

In the original series of cases reported by the Drs. Dick, about half the persons who had never had scarlet fever gave no reaction, and only about one-sixth gave a strongly positive reaction.

Subsequent series of tests have given similar results.

One important link in the chain of evidence connecting this test with scarlet fever is the effect of convalescent serum. When blood is taken from a person who has just had scarlet fever and allowed to clot, the clear portion of the blood which separates out is known as convalescent serum. This con-

valescent serum seems to contain some of the antibodies concerned in giving immunity to scarlet fever.

If instead of doing a skin test with the filtered and diluted streptococcus toxin alone, it is used after mixing with convalescent serum, a positive reaction cannot be obtained, even in those who have given them with the unmixed filtrate previously. This indicates that the minute amount of toxin present has been neutralized by the antibodies in the scarlet fever convalescent serum.

Moreover, if the convalescent serum in very small amounts is injected into a person who has shown a positive skin test, after a few injections no reaction can be obtained with the filtered toxin. Here again a protective action of the scarlet fever immune substances is the most likely explanation.

The value of this work is far-reaching. The streptococcus has been definitely incriminated as the cause of scarlet fever. The skin test enables us to tell those who are and those who are not susceptible. This is of great service, particularly in choosing nurses immune to scarlet fever for children's wards of hospitals and in determining those children who need special care owing to great susceptibility to the disease. Best of all, it has led us to as definite a method of conferring immunity as we now have in the toxin-antitoxin mixture for diphtheria prevention.

When relatively larger amounts of the streptococcus toxin are injected beneath the skin in three increasing doses, five days to a week apart, immunity to the disease can be given in most cases. The immunity lasts months and perhaps longer. Worth while results have been obtained in many places with this immunizing and so far as I know no adverse reports have appeared. The success of the immunization in a given person can be judged not only by escape from contracting the disease, which almost any one might do under favorable conditions, but by a change in the skin test from positive to negative.

This same change indicating immunity can also be brought

about by the subcutaneous injection of convalescent serum, as I have already mentioned. But convalescent serum can be obtained only if there is a patient getting well from scarlet fever who is willing to donate a portion of his blood for the welfare of others. Convalescent serum had also been used fairly successfully in treatment, but often was not available when needed.

The Drs. Dick are not the only ones who have been working on scarlet fever. Long before the World War the Russians had made some headway, and it is only fair to say that even now many of them do not accept the results of the Chicago investigators. In this country much valuable work has been and is being done, particularly by Dr. Dochez of the Rockefeller Institute.

Dr. Dochez inoculated horses with streptococci, together with some substance such as agar-agar or gelatin. He has produced by this means a serum similar to diphtheria antitoxin which gives very satisfactory results in the treatment of scarlet fever cases. Many experts prefer serum prepared by Dr. Dochez's method to that prepared by the Dicks'.

To sum up, we have learned the cause of scarlet fever and how to guard against it. We have a simple skin test which will show those persons who are susceptible to the disease and roughly the degree of their susceptibility. We have an easy method of immunizing the susceptible individuals, and the immunity apparently lasts for some months to a year or even more.

Moreover, very hopeful results have been and are being obtained in treating cases of scarlet fever with serum from horses immunized by streptococci.

All these definite, concrete achievements have followed rapidly the identification of the germ causing the disease, and emphasize once more the value of research work even in seemingly unfruitful fields.

CHAPTER X.

DIPHTHERIA.

If our belief is correct that diphtheria is preventable by use of the Schick test and toxin-antitoxin, and is curable in most cases by antitoxin, we have only our own inertia to blame for the prevalence of the disease. The American Medical Association has recently published a study of the deaths from diphtheria in cities over 100,000 population. Diphtheria is chiefly a disease of cities and the crowded sections of cities, so the mortality figures given in this survey probably represent the disease in its most severe form. The death-rates in this table are arranged in five-year groups, beginning with 1890–1894. It was in 1894 and 1895 that the use of diphtheria antitoxin, discovered by von Behring, became general.

Before I get too far, I had better give the table I am going to use for my text.

	1925	1920–1924	1915–1919	1910–1914	1905–1909	1900–1904	1895–1899	1890–1894
Boston . . .	12.1	20.2	26.3	20.0	26.2	53.7	83.9	112.2
New York . .	11.0	13.9	21.8	28.0	40.0	58.0	85.8	134.4
Fall River . .	11.6	25.5	23.6	24.0	34.4	50.1	43.8	46.9
Chicago . .	8.0	17.5	31.2	37.9	27.0	33.9	69.7	117.3
Denver . . .	14.8	23.2	26.7	10.2	20.8	29.6	27.3	130.2

These figures represent the average number of deaths from diphtheria per year for five-year periods for every 100,000 of population, except the first column, which represents the rate for one year, 1925, only.

The drop in deaths from 1890–1894 to 1900–1904 represents largely the work of antitoxin and improved quarantine methods. The decline until 1924, except in New York, is chiefly due to improved diagnosis and treatment. In New York the marked decrease in deaths in the last five-year period is due to the intensive use of the Schick test to detect those susceptible to the disease and their immunization by toxin-antitoxin injections. By 1925 the results of similar but belated campaigns in other cities are shown by the lowered death-rates.

This statement is not made with any intention of belittling the value of antitoxin, one of the greatest boons ever given to childhood. Luckily our generation knows but little of the terrors diphtheria—often known as membranous croup—held for the parents of thirty years ago. To have to watch without being able to help a child alternately struggling for breath and falling back exhausted, its face purple and puffy, its eyes haggard and terror stricken, is ghastly, the more so through the knowledge that at any moment death may come. And antitoxin, if not given too late, can transform this struggle against the choking hand of death into the quiet sleep of recovery. Yet there are antivivisectionists who would have had our children dying of diphtheria rather than permit the experiments on animals that made antitoxin possible, who oppose even now the efforts of scientists and doctors to deliver us from disease.

The high diphtheria death-rate of Fall River, for 1920 to 1924, practically twice that of New York, fits in well with the U. S. Census returns for 1920, which showed the infant mortality-rate there to be the second highest of cities over 100,000 population throughout the country, and the highest for cities of its class. However, the 1925 figures show great improvement.

Of course the deaths from diphtheria tend to fluctuate from year to year, but by taking five-year periods most of these variations can be ironed out, and the figures give us a more or

less accurate picture of our progress or lack of it. The striking feature of the New York statistics is that they show a steady and rapid decrease, and the death-rate is now less than one-twelfth of what it was thirty years ago. Now the general sanitary conditions and the crowding of population are considerably worse in New York than most places. How is it that New York is able to make a showing so much better than other cities not far away?

The reason is that New York woke up several years ago to the need some other cities are just beginning to realize. Diphtheria cure is not enough; we must have prevention of the disease. It took the loss of millions of dollars and many lives to convince us that sprinklers were better protection than the fire department for our large buildings. It has cost the lives of many children to bring home to us what our health authorities have been trying to make us see, that a little work to prevent diphtheria is far better than to depend on antitoxin to cure the disease.

Antitoxin is like the fire department in more ways than one. Some one has to give the alarm to bring the fire department; to make the diagnosis of diphtheria in order to give antitoxin. If the fire has gotten too much of a start, the department may be unable to get it under control. Just so if the disease has gone too long before antitoxin is given, it is of no avail. On the other hand, the protection given by toxin-antitoxin to those the Schick test has shown to be susceptible is always on the job, just as the automatic sprinkler is always ready to put out a fire.

Most parents have learned by now from those responsible for the health of their children in school what the Schick test and toxin-antitoxin, these two new weapons in the war against diphtheria, are, but it will do no harm to describe them briefly.

The Schick test, introduced in this country by Park and Zingher of the New York Department of Health, is a method of determining which people are immune to diphtheria and

which susceptible. It gives no immunity; susceptible individuals are immunized by injecting the toxin-antitoxin mixture.

The test is very simple. It depends on the fact that diphtheria toxin—the poison formed by the diphtheria bacilli—irritates the cells of the skin of people susceptible to diphtheria and has no effect on those immune to the disease. About four-fifths of those over five years old are found to be immune to the disease.

Two solutions are used. One consists of a very small amount of diphtheria toxin diluted in a weak solution of common salt. The other, called the control, is a similar solution heated to destroy the toxin.

A small drop of the diluted toxin is injected with a fine hollow needle between the layers of the skin of the right fore-arm. The object is to have the drop make a little blister in the upper layers of the skin, not going deep enough to draw blood.

The control solution is injected in the same way into the skin of the left fore-arm. If any change occurs here, the interpretation of the real test on the right arm has to be modified accordingly.

The picture of a positive test—meaning that the person has little or no immunity to diphtheria—is an area of redness $\frac{1}{2}$ to 1 inch across around the point where the injection was made on the right arm. This redness, which appears within forty-eight hours to thirty-six hours after the test, gradually increases up to the fourth day, with some swelling and itching or soreness. After that it fades gradually, turning brown and scaling a little as time goes on. The control injection on the left arm shows nothing.

If the test is negative, nothing at all can be seen on either arm.

This description holds for ordinary people, but some are "sensitive" to the protein of the diphtheria toxin. This sensitiveness is the same sort as asthma and hay fever. In these people the left arm as well as the right shows redness.

If the redness appears on both arms within twenty-four hours and begins to fade the third day, the test is negative. If the test behaves this way on the left arm, but reaches its height on the right arm on the fourth day and lasts considerably longer than the one on the left arm, it is positive.

The toxin-antitoxin is quite different. This is injected beneath the skin, where the body can absorb it. The principle depends on an observation made some years ago by Dr. Theobald Smith, then professor at the Harvard Medical School. He was immunizing guinea-pigs to diphtheria and found a higher degree of immunity and more lasting was given by diphtheria antitoxin if it had a small amount of toxin mixed with it than if the antitoxin was given alone.

When antitoxin alone is given it has no effect on the body and disappears quite rapidly. But if a little toxin is given with it, the antitoxin protects the body from harm while the toxin stimulates the cells to form antitoxin of their own. This "home-made" antitoxin is far more lasting and satisfactory than antitoxin injected into the body. The toxin-antitoxin injection gives an immunity that lasts a year anyway and usually much longer.

One of the places where toxin-antitoxin is of great use is in protecting those who have been exposed to a case of diphtheria. If given in time, it will nearly always prevent infection.

The increasing use of these two powerful aids against diphtheria ought to materially lower our mortality from the disease, and there is no reason why diphtheria cannot be stamped out absolutely in time. Antitoxin and quarantine measures seem to have brought down the diphtheria mortality as far as they can, and to lower the death-rate of this disease from 134.4 to 13.9 per 100,000 is no mean accomplishment.

It lies in our own hands now whether we shall continue to let our children die from diphtheria. Science has provided us with the necessary tools. It is up to us to use them.

CHAPTER XI.

COLDS AND SORE THROATS.

HAVE you figured out yet how you got your last cold? If so, you know more than all the scientists who have worked on the disease. The common cold—its very name suggests its prevalence—probably gives more discomfort and inconvenience than any other disease. Yet owing to its mildness and perhaps in part to its very commonness, only of late have research workers paid much attention to it.

When the study of colds was first begun, a germ called Micrococcus catarrhalis—about one-tenth the size of a red blood cell and shaped like a doughnut broken in two—was found in the nose and throat of people with colds. So it was believed to be the cause. But a little later some one spoiled the theory by discovering the same germ in healthy people. Then one by one the other bacteria that live in the nose and throat took their turn at being blamed for colds.

When all the germs revealed by the microscope gave out, the supporters of the infectious origin of colds took refuge in the filterable viruses—disease-producing substances of unknown nature, only a few of which can be seen by even the most powerful microscope, and which pass through fine filters that keep back practically all bacteria. The beauty of believing in a filterable virus is that our only sure test for its presence is the causing of disease. So one great source of criticism, that of its occurrence in healthy persons, is automatically ruled out.

Before I get too deep in the subject, it is just as well to define the term "cold." To a doctor, it means mild inflammation, with or without excessive secretion, of the mem-

branes in the nose, throat or bronchi. Tonsillitis is another disease definitely caused by the streptococcus germ and without relation to colds.

The familiar symptoms that nearly all of us have felt and some even now are feeling, are irritation, dryness, and swelling of nose or throat or bronchi, usually followed by excessive secretion of mucus, causing nasal obstruction, and a dry or loose cough. Along with these often go fever, headache or constipation.

The one thing every sufferer from a cold is well supplied with is advice. Nearly every one has a pet remedy, and knows just how to cure the cold and how the sufferer got it.

Drs. Jordan, Norton and Sharp attempted to find out how 2388 university students caught cold. Nearly 2000 were in the University of Chicago and 200 each in the University of Texas Medical School, Galveston, and the California Institute of Technology, Pasadena. Two colds a year was the favorite number in all three localities, and the great majority of colds lasted from one to three weeks.

Only 8 per cent of the Chicago students, 10 per cent of those at Pasadena and 23 per cent of those at Galveston averaged less than one cold a year. The nose was the favorite starting place, and one-third of the colds extended to the chest. So much for the colds.

How did these students think they caught cold? For convenience, Dr. Jordan divides the answers to his question in three groups—factors throwing strain on the heat-regulating mechanism of the body, such as chilling, exposure to draughts, wet feet, change of climate, and so on; those relating to contagion, and finally such causes as constipation, fatigue and over-eating.

Sixty-four per cent of the students believed some strain on the heat-regulating mechanism of the body was at fault—and just as many proportionately in the vaunted climate of Pasadena as in the windy city. Only 22 per cent believed they had caught their colds from others. More than one

philosopher has claimed science merely provides an explanation for popular beliefs. Will the history of the cold be another example of this?

But you may say, how is it that colds so often run through a family or school? Here we have groups of people living under the same conditions, so it is almost impossible to rule out exposure to chilling in favor of contagion.

Mudd and Grant, in the Harvard Medical School, rigged up an ingenious electrical device to record the temperature of the membranes of the nose and throat, and then chilled the body by draughts and in other ways. They found that whenever the body was chilled, the temperature of the throat was lowered and its blood supply was cut down, which gave any germs present a much better chance to get a foothold.

It may well be that this increased local susceptibility to the germs normally living in nose and throat will be the explanation we shall finally accept. It is difficult to see why chilling or draughts or wet feet should in themselves cause trouble in so protected a place as the inside of the nose or throat.

Another possibility is that the group of symptoms which we label as "cold" is not a single condition, but several. Some of the most carefully done of recent work, that by Olitsky and McCartney in the Rockefeller Institute, points in this direction. By washing out the nose and throat of people in the earliest stages of colds, they obtain a substance which when passed through fine filters could produce colds in healthy individuals. Yet there was one serious hitch. When the colds were those due to exposure to the elements or chilling, no virus could be obtained. Its only source was from those colds caught from other people. In other words, their work confirms from a totally different angle the results the students gave to Dr. Jordan.

The evidence makes us think, then, that we are dealing with colds due to germs, and colds due to exposure. The

young lady shod with satin slippers who picks her way through the slush on a down-town street in all probability has only herself to blame for the days of sniffling and coughing that may follow. And as likely as not the sneezing straphanger is spraying his neighbors with cold germs.

Both cure and prevention of colds depend on knowing the cause. Some pin their faith to vaccines made from one or another of the germs found in their throats, while others cling to red flannel underwear. Sometimes the believers get through a winter without colds—more often they do not. Any one method, followed narrowly, seems no better than another, and on close examination even the most enthusiastic devotees will admit their method fails once in a while.

The only one I have heard of that is absolutely sure is that of a skipper on Martha's Vineyard. He wears a sou'wester whenever he is outdoors, and never catches cold. Most of us would prefer an occasional cold to the sou'wester, however.

Owing to our present ignorance, the best we can do to keep free from colds is to avoid sudden exposure and keep away from people with colds. Even though a given person's cold is not contagious, it has no marks to distinguish it from the contagious type.

The attempt to cure a cold holds out but little promise. At present our remedies are flung out into the unknown, hoping that they may find and counteract the root of the trouble. Occasionally they appear to do so—more often they fail.

Though we cannot kill the snake, we can at least scotch it. When you feel a cold coming on, take a dose of physic and a bowl of hot lemonade and go to bed early. For the next few days eat lightly, drink all the water you can, avoid exposure and do not get overtired. Not a very spectacular course of treatment, nor will it produce striking results. But it will make life more bearable, and incidentally cut

down greatly the chances of such unpleasant complications as pneumonia or sinus infections.

Slightly related to a cold is sore throat, which usually takes the form of tonsillitis.

A week or two ago I had an attack. When one holds back from swallowing until the last possible minute, and has to grit his teeth to swallow at all, and when as well one is feeling pretty mean all over, if any thinking is done, it is apt to be about his disease. I found I knew the general treatment of tonsillitis and the usual outcome, but that was about all. So I decided to hunt up all I could find about the cause and prevention of the disease.

Nearly everyone who has worked on the disease agrees that it is caused by the invasion of the tonsil by a germ called Streptococcus hemolyticus, the same one we have already spoken of in the chapters on blood poisoning and scarlet fever, which when seen under a powerful microscope looks like a string of beads and which destroys, or hemolyzes, the red cells of the blood and furthermore breaks down the hemoglobin which the cells carry.

Ask any experienced bacteriologist which one of the common germs he would least care to have attack him. The chances are that he would name the Streptococcus hemolyticus. It is a "mean bug." Usually unable to invade the healthy body tissues, once it gains entrance to the blood through a cut or injury, or to the body cavity, as in child-bed fever, the chances of pulling through are none too good. That is, once it has invaded the body, it has things all its own way, but it needs help to get in.

In spite of its low invasive power, it apparently can get into the tonsils very easily. Luckily for us, it nearly always stops there, and does not set up blood poisoning.

The first day my throat was sore, I took a culture from my tonsils and found Streptococcus hemolyticus there in great numbers.

Now it is not very unusual or startling to find the strep-

tococcus in the mouth and throat, living there peacefully and doing no harm. In September and October, when streptococcus disease is not common, two doctors at Johns Hopkins in Baltimore took cultures from the throats of 175 people. Sixty-seven of them had had their tonsils removed. Only 9 per cent of these had streptococcus in their throats, and then only a few. But of the 108 who still had their tonsils, 37 per cent carried large numbers of streptococci, chiefly in their tonsils.

The question that comes up, do these people who carry streptococci in their throats ever get tonsillitis, and if not, why? Of course where the germ appears for the first time in an attack, it is easy to see that it is to blame. But if the people who normally have streptococci living in their tonsils come down with tonsillitis, is it because the boarders suddenly attack their host when he is tired or run down, or because he has picked up some new germs that have not learned to live in peace with him?

Drs. Bloomfield and Felty, who studied these 175 people, watched them from September to April. During this time, 39 came down with tonsillitis. The queer thing was that of the people who had streptococci living in their throats only 1 had tonsillitis, though just as much exposed to infection as the others. The other 38 cases occurred in those who had no streptococci previously in their throats or tonsils, and had therefore caught the infection from some one else.

Though having a germ which can cause tonsillitis as a steady boarder in your throat seems pretty risky, it is apparently safer than not having them. My own experience checks up well with these results. Only two weeks before my throat began to get sore, I had made a culture from it and noticed that no streptococci were present.

The next problem that presented itself was to find out whether the germs had established themselves in the tonsils without any fuss at all. By going back over the history of the carriers, 30 out of the 39 had had tonsillitis some time

previously. In other words, they had earned their immunity. Among persons immune to tonsillitis, there appear to be two types. The first group is made up of the ones we have just been talking about, the carriers. The other consists of those who seem to be naturally resistant to the streptococcus. Even though not having it present in their throats as protection, they do not get tonsillitis, even though repeatedly exposed. The reason for this immunity we cannot as yet figure out, so we label it "natural immunity" and let it go at that. Somehow giving a fact a name eases our minds, even though we do not know a bit more about it than before.

The natural immunity is much the better type to have, both for ourselves and others. If the carrier's germs leave him, his immunity goes too, and once gone, he is very susceptible to the disease. It is possible to think of the streptococci as conferring immunity by a sort of vaccination—their presence irritating the body just enough to make it defend itself by producing some antitoxic substance, which quickly disappears when the germs stimulating its production are no longer present.

Then too, the carrier is a risk, sometimes great and sometimes small, to those about him. If he coughs or sneezes, the chances are he will spray a few streptococci into the air, and they may light in the throat of someone not immune to tonsillitis. Just because streptococci live in peace in Smith's throat, there is no guarantee that they are harmless to me if they land in mine. It is hardly complimentary to a watch-dog to compare him with the streptococcus, but the simile may help. A watch-dog will not bite his master, but he may bite strangers. Just so Smith's streptococci may treat him well, but may attack me viciously.

So far as we know, the main supply of sore-throat germs comes from these carriers, who are relatively safe themselves but a source of more or less danger to others not immune, and from actual cases of the disease. As most people feel

sick enough to stay in bed or at least at home when having an attack of tonsillitis, the chief danger is from the carrier.

We know from numerous experiments with animals and from careful study of epidemics, that germs can increase their power of doing harm—their virulence. Also, people vary in the resistance they offer to invasion of germs. One of the best ways to increase the virulence of a germ is to pass it rapidly from one animal to another.

Smith has a nice tame family of streptococci in his throat. He sneezes and sprays a few out. Jones breathes them in, and they grow awhile in his throat, but do not make him any trouble. A day or two later Jones coughs and drives out a few of his boarders, say in a crowded subway train. Jackson breathes in a few, but he is immune, so nothing happens. But Harris also breathed in his share. He was tired and a little chilly and the streptococci got the upper hand. Next day it hurt him to swallow and he felt mean. That night he had a fever. In the morning the doctor came, said "tonsillitis," and gave him some medicine.

Of course instead of one healthy carrier like Jones, in real life there are probably dozens, but it works out in the same way.

For the sake of others, it is well to remember that your cold may be of the contagious type and that it is up to you to keep it to yourself. Generous and unobtrusive use of the handkerchief is a fairly good way to keep it from spreading. Sneezing and coughing spray out droplets of moisture loaded with germs and any one nearby is almost certain to get a good dose of them.

CHAPTER XII.

PNEUMONIA.

"Captain of the Men of Death," the phrase John Bunyan applied to consumption, is Sir William Osler's characterization of pneumonia. From time to time the disease is elbowed from its place at the head of the list of fatal diseases, but never for long.

We usually think of pneumonia as a disease of winter, going with cold and wet and exposure. The tragic death of President Harding brought home to us all its lurking treachery. Summer and winter the germs are present, merely waiting for fertile soil in which to develop. This soil they find in those exhausted by overwork or illness.

But pneumonia can strike down the robust nearly as readily as the feeble. The memory is still bitterly fresh with us of the ravages of bronchopneumonia combined with "flu," when the young and strong appeared to be the favorite victims.

The great group of pneumonia can be split into two main types, lobar pneumonia, the common acute disease, and and bronchopneumonia, which is likely to follow in the train of other sickness or attack weakened individuals. The lobar type is so-called because an entire lobe or more of a lung is attacked and put out of commission, while the bronchopneumonia is scattered throughout the lungs, spreading out into the lung tissue from the air-passages. Just what determines the type is uncertain. The same bacteria are often found in both, and looked at under the microscope, the picture of the disease process in the lung is practically the same.

If we are unlucky enough to get pneumonia, most of us are satisfied with knowing that we have it, or perhaps have ambition enough to ask the doctor whether it is the bronchial or lobar kind. If it is the lobar kind, though, it helps the doctor a good deal if he can find out which particular one of the four brands of pneumonia germs is making the trouble.

The microbe which is the cause of pneumonia is what bacteriologists call a diplococcus—which is simply the Greek way of saying "two berries." It is shaped like two soft-nosed bullets from 0.45 cartridges placed butt to butt, imbedded in a layer of jelly, and is about one-half the width of a human red blood corpuscle. The jelly which the germ secretes about itself is called the capsule, and serves to protect Diplococcus pneumoniæ—to give him his full title—from the attack of the fluids and white blood cells of the body when he gets in and tries to make trouble.

There are four sorts of these pneumonia germs, which all look just alike, except that one, named Type III, has more jelly around him than the others. The difference is not in their shape, but in their chemical properties. In order to test out these properties, bacteriologists use immune sera.

If an animal is made immune to any given germ by injecting small amounts of either dead or live germs of the same kind, the animal's serum, which is the clear fluid that separates from its blood on clotting, will make the germs bunch together, or agglutinate. So if we want to find out just what germ we are dealing with, we can try it out against these various "known sera," which are kept on hand in the laboratories, and the one which makes our germ agglutinate will tell us its name.

Suppose a doctor finds a germ which he thinks may be typhoid and wants to make sure. He grows the germ in broth, sterilized by heat, in glass tubes, and when he gets a cloudy fluid, due to the myriads of germs swimming about, he mixes a drop with the serum from a rabbit immunized to

typhoid. If it is a typhoid germ, a drop of the mixture looked at under the microscope will show the microbes bunched up together in motionless clumps instead of swimming freely around. Another test is to add several drops of the broth culture of the germs to tubes of the diluted antityphoid serum, and if the germs are those of typhoid, they will fall to the bottom in little clumps, leaving the fluid clear.

To return to pneumonia. There is one more peculiar thing besides shape about the pneumococcus, as the pneumonia microbe is often called. Bile will dissolve it. Add a few drops of bile to a tube of broth hazy with pneumococci and put it in the incubator at body temperature for an hour. Then look at it. The broth is perfectly clear, not a trace of the germs can be found, even with the microscope, and the fluid is perfectly harmless.

The brands of pneumonia germs are called Types I, II and III, according to their agglutination by known immune sera arbitrarily named. Those which will not clump together when treated by any one of the three standard sera, and yet dissolve in bile, are called Type IV. This Type IV, made up of a great many different races, is a sort of scrap-bag into which the bile-soluble diplococci not clumping with the three standard sera are thrown.

Type III, in addition to being agglutinated by Type III serum, has a larger capsule around it than any of the other types, and when grown in large masses has a peculiar sticky consistency.

In finding out the type causing any given case of pneumonia the first problem is, of course, to get the germ making the trouble. This is done by injecting a little particle of the sputum raised by the patient into a white mouse. White mice are especially sensitive to pneumonia, as the germs grow very rapidly inside them. In twenty-four hours the mouse is killed by etherizing, if not already dead from the pneumonia germs, and the fluid inside it is washed out. This is spun round in a centrifugal machine to get rid of the pus

cells present. The fluid which is left is loaded with pneumonia germs, descendants of those which were raised in the sputum from the patient's lung.

This fluid, cloudy with pneumococci, the bacteriologist uses to make his test. He has tubes containing sera of each of the three types and one containing bile. To each tube he adds a drop of the fluid from the mouse and puts the set of tubes away in the incubator for an hour.

If at the end of the hour, the tube with Type I serum is clear, with little flakes at the bottom, and the tube of bile is clear, while the rest are cloudy, the germ belongs to Type I; if tube II and the bile tube are clear, it is Type II; and if tube III and the bile tube, Type III. If all three of the serum tubes are cloudy, and the bile tube alone is clear, the germ belongs in the scrap-bag, Type IV.

Why bother with which of the four types is making trouble in any given case? In the first place, the patient's chances of getting well depend a good deal on the type causing the pneumonia. In a large series of cases, Cole of the Rockefeller Institute for Medical Research found that of those sick with Type I pneumonia 25 to 30 per cent died, with Type II, 25 to 30 per cent; with Type III, 50 per cent; with Type IV only 15 to 20 per cent. In the Boston City Hospital, the mortality from all types combined varies from 12 to 35 per cent, with the season and the various epidemics.

Another reason for finding out which type is making trouble is that there is a serum treatment for Type I. This is by no means a sure cure and is in no way comparable to the antitoxin for diphtheria or lockjaw, but still is worth trying in favorable cases of Type I pneumonia. This serum was first made and used at the Rockefeller Institute by a number of scientists, chief among whom were Cole, Avery and Dochez. While the results of the serum treatment seem promising, its value is by no means established as yet. A great deal depends on giving it early in the course of the disease, and one reason that the serum does not show

up well in the statistical returns is probably because it is sometimes used as a last resort and late in the disease.

The serum must be given with great care, and the patient tested out by having a few drops injected under the skin before the main dose, in order to be sure he will not react unfavorably to it.

The chances of getting any given type when you have pneumonia varies a good deal. Cole, in a series of 758 cases in New York, found that Type IV was the least common. The late Dr. Lesley H. Spooner of the Harvard Medical School analyzed 2980 cases in which the type had been determined. He found that 29 per cent were Type I, 20 per cent Type II, 11 per cent Type III, and 39 per cent Type IV. The weight of evidence seems to be that Type IV is the most common variety. This is a very good arrangement, as it is the least dangerous of all.

The queer thing about the pneumococcus is that most of us have it in our mouths and throats a good part of the time, and it never troubles us. Several other germs cause the disease, but their record is too low to bother with.

So we have the pneumococcus with us almost all the time, sometimes getting down into the lungs, when it may cause either lobar pneumonia or the bronchial type. What the determining factor may be is a mystery. Type IV is the commonest in the throats of healthy individuals, and seems to produce the mildest form of the disease.

However, no one type is a constant boarder. When I was a medical student, I took cultures from my throat from time to time, and usually found a pneumococcus Type IV to be present. After working for quite a while in a hospital ward where there were a number of patients with Type I and Type II infections, I found the Type I had moved into my throat and driven out the previous inhabitants. A couple of weeks later the Type IV organisms were again in control. I had been perfectly well all the time and could detect no difference in my throat when the races changed.

Once they get established in the lung, though, the type makes a big difference.

Pneumonia is no new disease. Hippocrates was well acquainted with it, and Aretaeus, a Roman physician, who lived some nineteen hundred years ago, has left a description of the disease that any modern physician might envy. At least one of the great plagues of the middle ages was in all probability a pneumonic outbreak. From the very beginning its successful treatment has baffled generation after generation of doctors.

I remember well the vivid impression it made on me when I heard Dr. Cabot say, some years ago, that study of the records of the Massachusetts General Hospital showed nearly as high mortality for pneumonia at that time as at the beginning of the nineteenth century. And no very great advance has been made since that time.

Few diseases are more dramatic than pneumonia. A healthy man suddenly taken with cough and pain in the side of his chest may in a few hours be delirious, gasping for breath, on the verge of death for days. Then, on the sixth or seventh or eighth day comes the crisis, and almost in a few minutes he may be well started on the road to recovery. Death may occur just as suddenly as the beginning of recovery, and not infrequently snatches its victim just as he seems to be out of danger.

The sudden recovery is often almost comic. One patient, I remember, was brought in by the ambulance in a state of semi-coma. He was taken to the ward, where the interne examined him, looked pretty blue and went away for the oxygen tank as a last resort. When he came back in a few minutes the man was weak and soaked in sweat, but grinned up at him and asked for a cigarette.

One of the characteristics of infection with the pneumococcus that makes the disease peculiarly hard to treat is that the germs are not content to stay in one part of the body, but invade the blood stream and may be carried anywhere.

This may not make trouble, but often does. The membranes of the spinal cord and brain may be attacked, giving a meningitis, usually fatal. The pericardial sac, in which the heart lies, may be attacked and filled with pus.

Research is constantly being carried on, trying to devise some way in which we can over come this "Captain of the Men of Death," but so far but little has been accomplished. What the future may hold in the way of cure or more successful treatment no one can say. But we can all of us protect ourselves more or less by keeping in good trim and avoiding so far as possible the sneezers, coughers, and spitters that are unconsciously scattering the germs.

CHAPTER XIII.

TUBERCULOSIS.

To those of us who have thought much about tuberculosis, or have seen it in our friends, it seems as if the disease must be inherited. Particularly in the smaller manufacturing towns where every one knows or at least knows of every one else, and where tuberculosis, or so-called "consumption" is particularly prevalent, instance after instance comes up where one generation after another has been scourged by consumption. Probably some of you who read this are suffering from the disease and remember that one or both of your parents had it.

Yet strong as the circumstantial evidence may be, though case after case be given where the disease has appeared in successive generations, the most careful scientific work has been unable to verify inheritance of consumption, and indeed, has proved that it cannot be inherited.

There is not much mystery about consumption nowadays. Luckily, we are almost all of us reached by one or another of the many agencies fighting the disease, and know a little about it. It is now forty-four years since Robert Koch discovered the germ which causes tuberculosis; a curved or slightly bent rod, about one-half as long as a red blood cell is wide. In those forty-four years great strides have been made in discovering what the germ does in the body and how it does its harm.

Hippocrates has left us an excellent description of consumption, as good today as when it was written. However, it was only a little over a century ago that Laennec, a French physician, laid the foundation for our present day knowledge

of the disease and gave us a practical method for the recognition of pulmonary tuberculosis. The last few decades are the ones in which progress has been made; progress due to four factors, and real progress as is shown concretely by marked reduction of the death-rate.

The four causes tending to decrease the amount of tuberculosis are: (1) The improved social condition, the higher standard of living, with better housing, better habits, and better food. (2) The realization by all of us that it is necessary to lead hygienic lives and especially to have some regard for the health of others, as shown by the decrease in spitting in public. (3) The cases of consumption are seen earlier and the condition recognized and treatment begun before it is hopeless. (4) Active cases of the disease are more apt to be in sanatoria and hospitals now, where they cannot serve as foci of infection, as they did formerly, when living and working in the community.

But to return to the inheritance of tuberculosis. By inheritance, or rather an inherited condition, we mean something, good or bad, which the unborn child has received from its parents. Since the mother is the one on which the developing embryo depends for life and growth, she is the most important one as regards inherited diseases.

Yet, even when the unborn child is in its mother's womb its blood does not mingle with hers directly. Instead there is a complicated interlacing mat of bloodvessels, some from the child, some from the mother, making up a mass of blood-filled tissue, the placenta. The interchange of food, oxygen and waste products takes place by diffusion through the thin walls of these tangled blood channels.

Experiments have been made again and again. Human cases have been watched and studied time after time. Never has a case been found in which tubercle bacilli have been able to make their way through the placenta from the mother's body to that of the child. In other words, tuberculosis is not an inherited disease.

Yet there must be some reason for its occurrence in families. Instances are too numerous, observations too careful, for the familial characteristic of the disease to be merely apparent, the result of mere coincidence. Where does the explanation lie?

In the first place, for the tuberculosis germ to grow, it must have fit soil. The parable of the sower is peculiarly apt to the present case. Some of the germs fall on stony ground—people with good constitutions or some immunity to the disease—and wither away. Some fall among thorns, that spring up and choke them—the protective agents of the normal healthy body. But some fall on good soil and bear fruit, increasing many fold. In other words, some people are peculiarly susceptible to infection with the tubercle bacillus. It is this tendency to the disease, and not the disease itself, which is handed on from parent to offspring. This hereditary tendency which provides fertile soil is one of the two main factors in the apparent hereditary transmission of consumption.

The other factor is of equal importance. No matter how fertile the soil may be, without seed there can be no growth. Nowhere better than in the intimate contacts of family life, particularly between mother and child, can the seed be sown on the soil prepared for it. Living in the same rooms, fondled, kissed, creeping about on floors where the tuberculosis germ may be lurking, the child of a consumptive has a pretty slim chance. No child is any too strongly resistant to tuberculosis, and the chance of an overwhelming infection in the home of a consumptive is great.

The droplets, far too often germ laden, that are spread broadcast by the cough of even a careful victim of tuberculosis settle everywhere on articles a child's busy fingers can carry to its mouth, or ones that it handles, loading its hands with infectious material. A child may escape infection once, or twice, or several times, but exposed week in and week out, its chances are pretty slim.

There is in many cases no reason other than the carelessness of its parents that the child of a consumptive should fall a victim to the disease. Of course in some cases the susceptibility, or hereditary tendency to the disease, is too great to fight. Here nothing can be done. But this should never be assumed to be the case. Let every child have a fighting chance.

Consumption occurring in generation after generation has no right to exist so commonly. It is only by virtue of ignorance and carelessness that it does.

It is only natural that in so common and so chronic a disease as consumption that many "cures" should be proposed, some honestly, as the substance prepared by Professor Dreyer in England, and some purely to extort money from the victims of the disease. But none are of much avail.

At present a gold-containing compound, "sanocrysin," has aroused much interest, especially in Europe. Some of the reports are very encouraging, but as yet it is too soon to judge.

Aside from the wide-spread occurrence of tuberculosis, its treatment is a fertile field for the quack and a pitfall for the honest practitioner, because of the spes phthisica—that pitiful sense of well-being that makes the consumptive think he is getting better when he is really going down hill. How misleading this may be was well shown in the early use of Koch's tuberculin.

Robert Koch was the discoverer of the tubercle bacillus, and his influence in medicine was second only to that of Pasteur. Consequently, when he announced that tuberculin, an extract which he had prepared from the tubercle bacilli, would cure consumption, the whole world was aroused.

A committee of American physicians went to Germany to investigate the cures and to learn the methods. They saw the cases, were convinced that the patients were improved. But one of the patients died—of consumption. And one case after another became worse. Spes phthisica was respon-

sible again. Who would not feel better, with the assurance of a cure from the discoverer of the cause of tuberculosis himself?

Then for years the field of tuberculosis cures was left to quacks. Not that the disease was not curable. The heroic work of Trudeau, the faithful care of the well-trained family physician, led the consumptives back to health by rest and open air and good food. But the cure is slow—the promises of the quacks, the testimonials (often still in use after the death from consumption of the sufferers who had written them) of patent medicines are enticing.

There is no short-cut road to health for the consumptive. Health he can regain if he is willing to look the facts in the face and do his best. Most cities and states have excellent sanatoria open to those who cannot afford individual treatment.

Some few may be cured by life in the Adirondacks or New Mexico; some few by collapse of the diseased lung to give it rest; some few by ultra-violet rays; but the sooner the consumptive and his friends and relatives realize he has a long road back to health, but a fairly sure one, the sooner he will stop wasting his money and courage in chasing false cures.

It may be some day we will have a quick and sure cure. There are many good scientists working for that all over the world. But until that cure is found and tried and offered with the full approval of the medical profession, we must stick to the slow but sure road which we know, the road marked out for us by such men as Trudeau and Lawrason Brown. Rest, fresh air, good food, together with such drugs or special methods of treatment as the attending physician finds necessary do not make a spectacular showing against the testimonials of the quacks—but most of the testimonials when we look closely are balanced by a death certificate.

However, tuberculosis is gradually being driven from its position in the first rank of killing disease. In twenty

years' time a great deal has been accomplished, though there is plenty more to do. In 1900 the death-rate from tuberculosis for the registration area of the United States was 195.2 per 100,000, lowered to 112 per 100,000 in 1920. One of the strongest indications of the progress we have made is that we are now working not only to save the lives of consumptives, but to find suitable employment for the cured cases.

The time was when we thought our job was done when the consumptive no longer showed signs of active disease. Now we are trying to see that he stays a "cured" or "arrested" case. Too often tuberculous patients discharged from hospitals or sanitaria have been obliged to engage in work far beyond their strength in order to provide for themselves or their families, with the result that the tubercle bacilli once more get the upper hand. In New York two factories are maintained to give the arrested cases of consumption suitable employment. One factory makes watches, the other clothing.

England has a government aided colony for tuberculous persons where they can earn enough to support themselves and many support their families.

There are a number of points that will have to be hammered home however, before we can make much progress along these lines. The first and most important is that heavy work is out of the question, for a long while at least. So the machinist or truckman cannot go back to his job; some light work for which he is or can be fitted must be found. With clerks or stenographers, of course, this problem does not have to be faced. Just because the sanitarium has put a dozen or so pounds of fat on a man and stopped his fever and cough, that man is not fitted to work his head off. He must go easy, for a good while at least.

Then the average employer has a prejudice against hiring an ex-consumptive. The danger of consumption and of its spread by spitting and coughing has been so thoroughly

taught we have a strong, and well worth-while, aversion to any possible source of tuberculous infection. Yet it is not fair to the consumptive discharged from a sanitarium to discriminate against him for this reason. If he were a source of tuberculosis germs, he would not be discharged and allowed to go to work. Moreover, in the sanitarium he has been taught to look out for the safety of others and is more careful about coughing, spitting, or sneezing than most of us would be.

If the ex-consumptive is to make a go of it, he must keep himself under the close supervision of a physician and follow orders. He is like an engine with a leaky boiler. With low steam pressure and a careful engineer it can get along all right.

That the game is worth the candle many a cured patient can testify. I have before me some figures from the Mt. McGregor Sanitarium of the Metropolitan Life Insurance Company. In the last ten years 1470 consumptives, employees of the company, have been treated there. Fifty-eight and four-tenths per cent were discharged as quiescent or arrested, and 27.6 per cent as improved. When only the early cases of the disease are considered, 96.6 per cent were arrested or improved, and 85 per cent are at work. These figures show not only the chance of recovery from tuberculosis, but the great advantage of early diagnosis and treatment of the disease.

Of the discharged cases at work, 88 per cent are earning more than when they entered the sanitarium, 4 per cent draw the same pay, and 8 per cent less. Decidedly a hopeful outlook.

While we are patting ourselves on the back for taking care of our consumptive, it is just as well to remember that there are far more of them than there ought to be. Tuberculosis costs us about $500,000,000 a year. It is probably safe to say that very few of us get through life without exposure to the germs of tuberculosis. Whether we get consumption or not depends largely on our resistance.

In industry the important factors producing disease are not the direct ones, but the indirect. For every case of lead poisoning, "spelter shakes," or caisson disease, large numbers have fallen prey to consumption, pneumonia and other diseases largely through lowered resistance. The factors bringing about this lowered resistance can be divided into two groups. One is economic—wages compatible with only a low standard of living or irregular employment; the other hygienic—sanitation of the workroom, hours of employment, and so on.

A very considerable amount of work has been done on the relation of tuberculosis to various occupations. The following table summarizes a few of the more important occupations

Occupation.	Per cent of total deaths due to tuberculosis.
Coal miners	5.8
Bankers	5.9
Farmers	6.6
Textile workers	22.0
Garment workers	27.8
Teamsters	29.4
Glass-blowers	36.3
Hatters	36.6
Polishers and grinders	43.0

From these figures, some of which are eight or ten years old, unfortunately, and so probably much worse than they would be in those same trades today, it can be seen that tuberculosis mortality increases with poorer working conditions and particularly in dusty trades. The low rate of coal miners is partly artificial, as the accident death-rate is high.

What improvement of working conditions in the dangerous trades can do is well shown in the case of Solingen in Germany, which produced a good share of the world's cutlery before the World War. At Solingen in 1910, under almost ideal working conditions, out of every 1000 grinders and polishers 4.4 died from consumption. At Sheffield the

same year 15.1 died out of every 1000 grinders and polishers. Solingen had taught its employees not to spit, had cut dust and dirt down to a minimum and had good ventilation. Sheffield was dirty, smoky, and unhygienic.

New Jersey has given us a good example of what can be done by factory sanitation and instruction of employees in personal hygiene.

In seven of the most dangerous trades from the point of view of tuberculosis, hatters, stone cutters, metal grinders, moulders, founders, casters and plumbers, there has been a substantial decrease of tuberculosis. In one, the textile industry, there has been no improvement.

We are gradually learning to prevent tuberculosis. When proper care is given to the disease in its early stages it can be cured; the earlier the diagnosis the better the chance. The arrested case is being helped more and more to find its proper position in industry.

CHAPTER XIV.

DIABETES.

In the registration area of the United States the death-rate of diabetes has increased six times in the last forty years, while the death-rate from all diseases is dropping. (By registration area is meant that portion of the country where sufficiently accurate vital statistics are kept to warrant conclusions being drawn from them. The area includes about 80 per cent of our population.) This great increase in deaths from diabetes has been recognized and commented on for a good many years, and was mentioned in the census bureau reports as far back as 1906.

There have been two temporary drops to the climbing diabetic death-rate. One was from 1898 to 1901, following the influenza epidemic, which killed many people suffering from diabetes and who would otherwise have died from that disease. The second drop began with a halt in the rapid increase of the disease in 1916 and 1917, and the drop came in 1918, 1919 and 1920. Here again outside factors were at work. The years 1916 and 1917 were years of restricted diet, due to wartime conditions, and diabetes is more or less closely related to diet. The drop in 1918 was due to the same cause as the earlier one in 1898—the influenza epidemic. Both these drops were followed by very rapid increase in the death-rate.

The increase in deaths from diabetes has been most marked in those persons forty-five years old and over. In the later years of life women are much more commonly affected by the disease than men, though in childhood and youth males are more often the victims.

In the case of New York City we have accurate statistics on diabetes since 1866 and the same tendency appears there as for the rest of the country. The proportion of deaths from diabetes in those under twenty years has stayed about the same, while the death-rate for those forty-five years or over has increased more than twelve times.

But you say that the chances are more cases of diabetes are recognized by doctors now than formerly, and that therein lies the explanation. This is partly true, but not entirely, and certainly not enough so to explain the greatly increased death-rate. Diabetes ever since it was described by the Roman physician Celsus has been recognized by doctors for nearly two thousand years.

The chronic character of the disease, lasting for years, together with its distinctive symptoms, make it easy to recognize and generally bring the sufferer to a physician. The large quantities of sugar-containing urine, the thirst and hunger of the sufferers, their increased susceptibility to infectious diseases, are all very striking.

What is diabetes, anyway? At bottom it is a more or less complete inability of the body to use sugar and, to a lesser extent, fat. As all starchy foods are absorbed as sugar after being digested, this means that they cannot be properly utilized by the body. Since sugar is absorbed to a greater extent than it is used, it means that an abnormal amount is present in the blood.

When a substance reaches a certain concentration in the blood, it appears in the urine. The kidneys may be compared to a dam that holds certain substances in the blood. But just as water pours over a dam once its level in the basin above is high enough, any increase of substances in the blood above the level at which the kidneys hold them back means that those substances will appear in the urine. So when the sugar in the blood reaches a certain point it spills over the barrier of the kidneys and appears in the urine.

The work of Banting, the discoverer of insulin, and many

experimenters before him, proved that absence of a secretion from the pancreas caused diabetes. The pancreas is a gland about the size and shape of a banana that lies below the stomach. It has two functions. One is to make digestive juices, which are conveyed by ducts to the upper part of the small intestine. The other is to make insulin.

Insulin is manufactured by clusters of cells scattered through the pancreas which look quite different from the cells which secrete the digestive juices and which have no ducts. The secretion, therefore, is not carried to the intestine but is absorbed directly by the blood stream. This type of gland is known as ductless or endocrine.

When a sufficient amount of insulin is present in the blood, sugar is utilized normally. But when the supply runs low diabetes appears.

In the earliest stages of diabetes the blood sugar will be increased before it has spilled past the dam made by the kidneys and appeared in the urine. So to detect very early diabetes—and the earlier it is caught the more satisfactory treatment is—a test of the amount of sugar in the blood is advisable.

By careful dieting and cutting down of sugars and starches to the lowest possible limit, a diabetic could get along fairly well. The possibilities of this method were pretty well explored when insulin was discovered.

Diet must be controlled as carefully as ever with insulin treatment, however, because too much insulin for the amount of food eaten is just as dangerous as too little. Insulin keeps the level of sugar in the blood down toward normal limits and so renders the diabetic less liable to infectious diseases.

But even though we have figured on these steps correctly, why do the cells which make insulin, or to put it more accurately, control the use of sugar, go bad? In other words what is the cause of diabetes?

In a number of cases of diabetes, careful microscopic study

will show diseased cells in the islands of the pancreas. Yet some of these cells, with the methods of study we have available, appear to be normal.

The most commonly accepted explanation is that we eat so much starch and sugar, that these cells are over-strained and are permanently damaged. Though statistics are dangerous to rely upon, they give strong supportive evidence of this theory.

Diabetes is a rare disease among those doing hard manual labor, and very common comparatively in those who are over-weight and follow a sedentary occupation. It is especially common in well-to-do people who live well and exercise little. The per capita consumption of starchy foods and of sugar has increased greatly, and probably is a fair indication of a change in the average diet, protein being replaced to a considerable extent by carbohydrates. The annual per capita consumption of meat in this country has fallen in fifteen years from 179 to 165 pounds and that of sugar has increased to 100 pounds.

Probably the increased use of sugar is not itself the root of the evil, but a straw which shows the tendency to luxurious living.

The death-rate for diabetes is low among negroes who mostly are engaged in manual labor, and high among Jews, many of whom do little physical work and become obese.

Moderation in eating and exercise are not only of value in avoiding diabetes, but in keeping the general health in good condition. So whether time proves or disproves the theory that over-eating is an important factor in causing diabetes, avoidance of over-eating is a good thing.

The sooner a diabetic puts himself under the care of a doctor the better off he will be, and one of the valuable services rendered by periodic health examinations of middle-aged persons is the early detection of diabetes.

The one bright spot in all this spreading cloud of diabetes is the success of the insulin treatment. The death-rate

has begun to drop in the last year, coincident with the widespread use of insulin, which strongly suggests insulin to be the cause of the improvement.

Whether it will ultimately have any effect on the death-rate we cannot tell as yet, but we do know that patients treated with insulin and carefully controlled diets are apparently greatly improved.

The diabetic patient is like an automobile with faulty ignition. The engine is all right, the gasoline and air reach the cylinders, but there is no spark, and when the engine is turned over the mixtures go out unused in the exhaust just as sugar does in the urine of the diabetic. Dr. Banting's insulin is the spark the diabetic needs to ignite his mixture and set his engine to running properly.

Thanks to the labors of Dr. Joslin and Dr. Allen, careful dietary and hygienic control had lengthened the life of the diabetic to nearly six years, part of which were fairly worth living from the point of view of a healthy person, and had made those six years far more comfortable than had been dreamed possible twenty years ago.

Today Dr. Banting and his collaborators have given us a substance, dangerous, to be sure, as is any powerful drug, by the use of which we know the diabetic can almost always be rescued from the brink of death, even in coma cases which were formerly fatal in practically every instance. We know the patient can live a fairly active and comfortable life without feeling the pinch of hunger every minute and dreading blood poisoning from every scratch—insulin is a drug which so long as taken will in all probability keep the disease at bay.

Dr. Banting was not the first to try extracts of pancreas in treating diabetes, but the extract he used was free from the powerful digestive juices of the gland which had rendered useless earlier attempts.

Insulin is now being produced in sufficient quantities to supply all those who need it. The important and peculiar

feature of insulin is that it is not a medicine which can be taken a few days, weeks, or months and then cure the disease. Insulin must be taken as long as the diabetic condition lasts, which is the same as saying as long as the patient lives.

Now even if quinine cost $10 a dose, almost anyone with malaria could afford to take enough to be cured, for once cured he stays cured.

But with insulin we face a different problem. Insulin costs the patient using it anywhere from 2 cents to $2 a day. Does the good from the treatment offset its expense?

Its value, of course, was realized from the beginning. But whether this value could be expressed in dollars and cents was another question. In other words, is it fair to start patients on insulin when it might subject them to a severe financial drain? The Peter Bent Brigham Hospital set out to answer this question. It selected 5 cases and studied them carefully, before and after they had been treated with insulin, not only from the medical but the economic side.

This is what $250 worth of insulin did for one man. He was a business man, aged thirty-six years. He had had diabetes for three years. He could still work, but not much. He was gradually going blind from cataract in both eyes. No surgeon would operate, for his diabetes made the risk too great. He was started on insulin ten months ago. At once he began to improve, after three years of steadily going down hill. He was operated on and his eye-sight saved. For the last three months he has been working as well as he did before he got diabetes. Did he get his money's worth?

Another case was that of a waiter. He had been treated by the best dietary methods. He was still able to work, but was not a very good waiter, owing to his disease. His pay was $100 a month. After spending $10 a month for insulin, he felt a great deal better, gained strength, and earned $125 a month.

These cases will serve as examples of the others.

But do not think from this that every diabetic should have insulin. Dr. Joslin believes only severe diabetics actually need insulin. For the rest it is a luxury. It is very easy to say, "I will eat that second piece of bread and make up for it by an extra unit of insulin." But the first thing the patient knows, he will be eating more than his insulin can take care of.

The diabetic being treated with insulin must be just as careful in his diet as the diabetic under treatment with diet alone. The balance between insulin and food must be kept exactly. On the one hand lies sugar in the urine and diabetic coma; on the other, convulsions and even death from too much insulin.

But the most important point is to keep the urine sugar-free. The appearance of sugar in the urine, even in small amounts, is a danger signal that must not be disregarded. Either the diet must be cut down or the amount of insulin must be increased.

In other words, the diabetic, even with insulin, cannot abandon his Spartan life.

Yet diet is not the only thing diabetics must watch. From 1895 to 1913, 23 per cent of the diabetics who died in Boston hospitals died from gangrene. Since insulin has come into use death from coma is much less frequent, but death from infection and gangrene is almost as common, now making 45 per cent of the hospital deaths. Ninety per cent of the cases of gangrene occur in those over fifty years of age.

Progress now must be made in cutting down the deaths from infection and gangrene. As Dr. Joslin says, diabetes is not a mouth disease, but a foot and mouth disease. In the New England Deaconess Hospital, where diabetics get as fine care as it is possible to give, there is one nurse whose duty it is to look after the patients' feet.

To avoid gangrene, Dr. Joslin gives the following rules

which all diabetics should follow, and particularly those over fifty years of age.

1. Wash feet daily with soap and water. Dry thoroughly, especially between toes.

2. When thoroughly dry, rub well with hydrous lanolin, as often as necessary to keep skin soft, supple and free from scales and dryness. If nails are brittle and dry, soften by soaking in warm water one-half hour each night and apply lanolin generously under and about nails and bandage loosely. Clean nails with orange wood sticks. Cut nails straight across and avoid injury to the toes.

3. Wear shoes which do not bind or rub. Wear new shoes one-half hour only on the first day and increase one hour daily.

As regards cuts and breaks in the skin, he advises:

1. Thorough cleanliness with soap and water.

2. Avoid strong irritating antiseptics such as sulphonapthol and iodine.

3. Cover with lanolin on sterile gauze under a light bandage. Sterile gauze in small sealed packages can be purchased at drug stores.

4. Avoid using the foot as much as possible until wound is healed.

5. Consult your doctor for any infection.

In diabetes, cleanliness is pretty close to life itself.

Insulin works—and is a paying investment financially and medically. It is a necessity for severe diabetics—a luxury for mild ones. Insulin has cut down tremendously the death-rate from coma. The carefulness of the patients themselves must cut down the death-rate from infection and gangrene.

It was on January 10, 1922, that Dr. Banting gave insulin to his first 3 diabetic patients. Now thousands are being treated and its value is thoroughly established.

CHAPTER XV.

HEART DISEASE.

Few diseases are of greater importance than those grouped under the name of heart disease. In 1920, one-ninth of the total deaths occurring in the registration area of the United States were due to this cause. Almost all of us know of one or more sufferers from heart disease among our friends.

Trouble with the heart may be of two sorts—functional, where there is temporary or slight interference with its normal action, and organic, where there is permanent structural change.

The heart is a four-chambered pump which keeps the blood in circulation, the right set of two chambers receiving the blood from the veins and pumping it to the lungs, while the pair on the left side receive the blood from the lung and pump it out to the body. The two chambers which receive the blood are called auricles, those which force it out are called ventricles. There is a valve between each auricle and ventricle and also between each ventricle and the vessel into which it forces the blood.

Thus there are two chances for trouble in the heart—one if the valves get out of order and one if the muscle is damaged. The common type of heart disease in children and people up to forty years of age is valvular trouble. Inflammation of the valves may so distort them that the opening is too small or else that perfect closure is impossible. In the first case the blood backs up behind the obstruction, in the second it leaks back through the defective valve. Either way it throws a greatly increased load on the heart.

When the heart goes bad in people over forty years of age,

it is usually because the muscle has been injured. This injury is generally due to poor blood supply to the heart muscle.

Few if any engines could do the work that the heart does. It pumps away about 70 or 80 beats a minute, from some months before we are born until we die. It can increase its output five to six times almost instantaneously. Up to a certain point, the more work it has to do the better it does it.

The way the heart responds to increased loads thrown upon it is to dilate, permitting more blood to enter the chambers and giving the muscle fibers a longer distance and therefore greater chance to contract. If the extra work is long continued, the muscle fibers themselves enlarge, just as they do in our arms when we do heavy work.

A large part of the training process that athletes undergo is for the purpose of building up a reserve power in the heart, so that when it is called upon to do a great amount of work it can respond to the need. This is particularly true of those sports for which much endurance is required. The muscular efficiency of a man is measured by the extent his body will respond to increased exertion. The average normal man by an extreme effort can increase his muscular work and the rate of his circulation 600 per cent. This of course is due somewhat to the reserve power of the muscles, but the more work the muscles do, the more blood the heart has to supply them.

This 600 per cent margin can be much increased by training in the sense of gradually increasing exercise and well chosen diet. The heart in the trained man can greatly increase its output of blood over the normal amount without making the beats faster. The average man has to get such an increased output chiefly by speeding up his heart beat, and when his heart is giving its maximum output, that of the trained athlete is only beginning to speed up. Moreover, it takes far longer for the rapid pulse of the "soft" man to come back to normal after exercise than it does that of the trained athlete.

The so-called "second wind" in severe muscular work or hard exercise is the result of the heart adjusting itself to the load thrown upon it. So long as the blood forced into the circulation is less than the muscles need, we have the feeling of being "all in," but once the heart has gotten its output of blood equal to the demand, the sensations of breathlessness, pain and exhaustion disappear.

Of course the heart muscle itself needs a generous supply of blood to keep it going, and also a means of increasing that supply when it has a greater amount of work to do. There are two blood supplies to the heart muscle. One is a small system of vessels running from the inside of the heart into its wall, while the other and chief supply is the coronary arteries and their branches. These two coronary arteries receive the blood from just behind the aortic valve, where the blood-pressure is at its greatest. The blood supply to the heart muscle is much increased when the heart works hard, just as we give our motors more gas when we want more power. When the heart is working its hardest, the coronary vessels may carry ten times as much blood as they do when the body is at rest.

Anything which lessens the amount of blood the coronary arteries can bring to the heart muscle will result in its malnutrition and consequent damage. Rarely harm may be done to the muscle by direct injury, as by the poison of diphtheria, prolonged malnutrition, direct invasion by germs, or phosphorus poisoning. However, by far the commonest cause of injury to the heart muscle is hardening of the coronary arteries, the same process as occurs in other arteries, usually associated with high blood-pressure, with consequent death of muscle fibers from malnutrition and their replacement by scar tissue.

Not infrequently a diseased coronary artery has become so narrowed that the blood flows through it with difficulty and finally clotting occurs. This usually leads to sudden death as the heart muscle is cut off from its blood supply.

A blood clot broken off from some other place and lodged in a coronary vessel has the same effect.

If death does not come at once, the portion of muscle supplied by that artery dies. Sometimes a scar forms, and sometimes the blood bursts through the dead muscle, resulting of course in death. This latter case is the only one I know of in which one finds a broken heart, and it is very unusual, writers of fiction to the contrary.

Nearly every one has exercised to the limit at one time or another and felt faint or nauseated with pain over the heart. This is due to the inability of the heart to meet the demands made on it. The same feeling, with the pain accentuated, is the one which warns the person suffering with damaged heart muscle that he has reached the limit of his strength.

As a general rule, pain is the outstanding symptom of angina pectoris, as heart injury of the type often associated with diseased coronary arteries is called. Whether the pain is caused by the diseased muscle itself or by the increased blood-pressure stretching the wall of the aorta—the great vessel which carries the blood from the heart—is an undecided point. In any case, it is a warning of an excessive load on the heart and is relieved by rest and lowering of the blood-pressure by such drugs as the nitrites. Even an emotional disturbance can bring on an attack of the pain. Hunter, the great English surgeon, who was quick-tempered and a sufferer from angina pectoris, once said, "I am at the mercy of any fool who makes me mad." Death usually occurs in one of the attacks.

In contrast to the diseases of the heart muscle are those of the valves. These, usually found in younger persons, are caused by germs. When valvular heart disease develops fairly late in life, the germ of syphilis is commonly responsible and attacks the aortic valve by extension from the aorta itself. One of the commonest causes of valvular heart disease is rheumatic fever. As yet we do not know just what germ is responsible for this disease.

Let us follow through a typical case of rheumatic fever. A youngster of ten has had a severe attack of tonsillitis. One day he feels slightly chilly, has a sore throat, furry tongue and fever. Next day the fever may go to 102° or 103° F., with heavy sweats, and his knee-joints are hot, swollen, tender, and very painful to move. After a day or two the knees begin to get better, while the pain and swelling appears in the wrists. A number of joints may be attacked, usually trouble beginning in one as the preceding one starts to clear up. The slightest motion of the affected joints causes intense pain. This condition may last for two or three weeks or even much longer.

The heart usually has been attacked at the same time as the joints. Warty growths form on the edges of the valve leaflets, so they cannot close properly and blood leaks back. This puts an extra strain on the heart muscle, which itself has been injured by the virus. Death may occur at this time, or healing may occur. When the process quiets down, however, the valve leaflets are left shrunken and deformed, thus permanently embarrassing the circulation of the blood.

Our methods of treatment of rheumatic heart disease are not very satisfactory. Rest in bed during the attack and for weeks or even months afterward and large doses of salicylates are the best remedies at our disposal. In heart disease particularly prevention is far more important than treatment. Care of infected tonsils and decaying or abscessed teeth is of great importance. Proper food, plenty of fresh air, and well-balanced exercise and rest are also essential. In susceptible individuals exposure to cold and wet may bring on an attack of rheumatism.

Every case of rheumatic fever is potentially one of heart disease, and even if no evidence of heart trouble appears, prolonged rest in bed and very gradual resumption of activity is the only safe rule.

Another germ causing valvular heart disease is the Streptococcus viridans, which grows slowly on the valves and

gradually distorts them. Diseased tonsils are thought to be one of the commonest routes by which this germ gains access to the body.

Now all these injuries to the valves throw more work on the heart muscle. Either the valve is narrowed so the muscle has to work extra hard to force the blood through, or so distorted that the blood leaks back again. High blood-pressure, which we will consider in the next chapter, also means more work for the heart.

The heart in disease responds to greater strain just as it does in health; it dilates, increasing the amount of blood it can hold, and its muscle hypertrophies, so that its power of contraction is increased. In addition, the number of contractions per minute is frequently increased. Thus we have a heart larger than normal, and with thicker walls than normal.

Ordinarily the hypertrophied heart can keep enough blood in circulation, but it is working full force all the time and when extra strain is laid on it, it has no reserve power to fall back on. Consequently it stalls under the excessive load and the circulation of the blood is slowed down, with over-filling of the veins. Dropsy appears and becomes more and more marked, fluid collects in the lungs, and death soon comes if the heart cannot be helped by proper stimulation and the extra load which brought on the decompensation cannot be removed.

Every one suffering from heart disease should determine the degree of activity they may safely employ and not exceed it. Shortness of breath is a fair indication that the heart is being over-worked. In addition, any sign of dropsy, such as swelling of the ankles, is a danger signal. A test much used by doctors is the determination of the so-called "vital capacity," the amount of air that can be blown into a spirometer with a single breath, as deep as possible.

Fortunately, we are learning more and more how to avoid trouble in heart disease, and even are making a little headway in preventing it.

CHAPTER XVI.

HARDENING OF THE ARTERIES AND HIGH BLOOD-PRESSURE.

Hardening of the arteries is so general a condition in elderly people that some scientists regard it as a normal accompaniment of old age. It is not restricted to man, for many of the herbivorous animals develop it, especially horses and cattle. Curiously enough arterial changes of this sort are very rare in meat-eating animals.

Though we hear a great deal of the relation of arteriosclerosis to the drive and nervous strain of modern life, even Egyptian mummies of King Tut's time and earlier have been found with hardened arteries. Since the disease has first been observed a vast amount of time has been wasted in discussion as to its cause. Alcohol, over-work, tobacco, over-eating especially of protein food, coffee, acute and chronic infections have all come in for their share of blame, and the ultimate verdict is the old Scotch one, "Not proven."

The walls of the arteries consist of three layers. The innermost, next to the blood, is very thin. This is called the intima. The next layer, the media, is made up of elastic tissue, smooth muscle cells and connective tissue. It is this coat that gives the arteries their necessary strength and elasticity. Outside these first two layers is a loose meshwork which contains small vessels carrying the blood essential for the nutrition of the arterial wall.

In one type of arteriosclerosis, the kind due to syphilis, destructive processes take place in the media and the normal muscle and elastic tissue is replaced by inelastic scar tissue. This variety is comparatively uncommon, however.

The first change that can be seen in hardening of the arteries of the ordinary sort is a slight patchy thickening of the inner layer, the intima. About the same time the elastic tissue in the media frays out to a considerable extent, so a good deal of the tough, rubbery quality of the wall is lost. Then degeneration of the cells in the thickened intima occurs and the products of the break-down of the cells attract lime salts from the blood into the vessel wall. Eventually bony plates may practically line the vessels.

As a result the arteries are converted into practically rigid tubes and their capacity is decreased by the thickening of the wall. Thus to maintain an adequate circulation, the blood has to be driven through the vessels at higher pressure. There is a kind of "eternal triangle" in our bodies, with the blood-pressure, the arteries and the kidneys at the angles. Any one of them may start to make trouble and drags the other two after it. Arteriosclerosis leads to high blood-pressure and kidney disease. High blood-pressure leads to kidney disease and arteriosclerosis. Kidney disease leads to arteriosclerosis and high blood-pressure. It will be helpful in understanding the problem to consider high blood-pressure at this point. Kidney disease we shall take up in the next chapter.

Hypertension, as high blood-pressure is known to medical men, is one of the ailments which has gained more and more prominence with the advance of modern medicine, and perhaps due somewhat to the increased wear and tear of modern life. In order to understand what high blood-pressure is, it helps to review the circulation of the blood, which most of us have forgotten since our grade-school lessons in physiology.

The heart is a double pump, as we said in the last chapter. The right side receives the blood, which drains from the great veins and pumps it to the lungs. The left side of the heart takes the blood as it comes from the lungs and pumps it out into the aorta, or main artery. From the aorta many branches are given off, becoming finer and finer,

until finally the blood reaches the hair-like capillaries. Through these it travels to the veins, which collect to larger and larger trunks, and eventually empty into the right heart in two great vessels. That is, the heart is pumping the blood through a closed system of tubes which spreads throughout the body.

At every beat of the heart, blood is forced out into the arteries, momentarily raising the pressure there. This increase of pressure is rapidly taken up by the elasticity of the walls of the arteries. This rise and fall of pressure due to the heart beat is what we feel as the pulse.

In a closed system such as the heart and bloodvessels there are two ways in which the pressure can be altered; one a change in the total volume of fluid in the system, the other a change in the system's capacity. In our bodies, ordinarily the kidneys guard against any increase in the volume of the blood. However, the small arteries are controlled by very sensitive nerves, which can change the size of the arteries they innervate.

Essential hypertension is the ordinary high blood-pressure, not caused by kidney disease or hardening of the arteries. It generally develops in the fifth decade of life, and is more likely to occur in those who are fat.

It is perhaps due to a nervous spasm of the smaller arteries, which close down and decrease the capacity of the bloodvessels, as well as offering more resistance to the flow of blood due to their smaller bore. This means that the heart has to maintain a higher head of pressure to circulate enough blood for the nourishment of the body.

We did not hear much about high blood-pressure twenty or thirty years ago because doctors did not have any ready way to measure it. They could only guess at it by feeling the pulse. It is only lately that physiologists have perfected the sphygmomanometer.

This instrument in its usual form consists of a rubber cuff which can be inflated, connected by a tube with a column

of mercury. The cuff is fitted around the arm above the elbow, and blown up with a bulb until it compresses the artery of the arm. The air is then gradually let out by a needle valve, and the height of the column of mercury in the tube is read when the pulse can first be felt, or when it can be heard at the elbow with a stethoscope. This is the systolic pressure, or pressure at which the heart forces blood into the arteries. When more air is let out of the cuff, the sound heard with the stethoscope changes, and the height of the mercury column is again read. This is the diastolic pressure, or pressure maintained in the arteries between heart beats.

A blood-pressure of 125 means that the pressure of a column of mercury 125 millimeters (5 inches) high is needed to blot out the pulse.

In order to know whether a given blood-pressure is high or not, we must know the normal ranges. In the following table, the average blood-pressures for various ages are shown, as determined in many thousands of normal people:

Age.	Systolic blood-pressure.	Diastolic blood-pressure.
15 to 19	120	85
20 to 24	122	85
25 to 29	123	85
30 to 34	124	85
35 to 39	126	85
40 to 44	128	85
45 to 49	130	85
50 to 54	132	90
55 to 59	134	90
60 to 64	135	90
65	136	90

These figures, of course, are only averages, and a wide range of variation is normal. In general, any blood-pressure up to 150 millimeters of mercury is not considered abnormally high and any of 160 millimeters or over is considered diseased. In sleep the blood-pressure is from 20 to 25 millimeters lower than when we are awake.

The blood-pressure is the result of the action of the heart,

and any increase in blood-pressure throws more work on the heart. When the heart has to do more work it has to enlarge, and after it enlarges up to a certain point it is hampered by its increased size and weakens. When an extra strain is put on the arteries for any length of time, their walls become diseased and hardening of the arteries results. Hardening of the arteries—for the body is like the house that Jack built—goes on to injury of the kidney and the brain.

High blood-pressure is more than the reading on a column of mercury. There are usually troublesome symptoms that go along with it. Perhaps the commonest is a headache, which begins when you first wake up and steadily gets worse until breakfast time. After breakfast it usually goes away for the day, though at times it may last for several days, always worse in the morning. Dizziness is another common difficulty and often trouble in speaking or weakness in one or more limbs goes along with it, lasting for a day or so. Nose bleeds are very common.

When the high blood-pressure begins to affect the heart, shortness of breath becomes noticeable, and the heart may flutter and throb unpleasantly. Occasionally there may be knife-like pains over the heart, which come suddenly and radiate to the back or shoulder. More rarely there is swelling of the legs.

With its almost inevitable companion arteriosclerosis, there is always the possibility of serious trouble from high blood-pressure. Cerebral hemorrhage, or apoplexy, is the usual end for victims of this disease. The blood under high pressure tends to break through the weakened walls of the hardened arteries, especially in the brain. A stroke of apoplexy, if not fatal at once, usually results in some degree of paralysis, though the extent of damage done may vary greatly.

Suppose you have too high blood-pressure. What can you do about it? The first thing to do is to put yourself under the care of a physician. He will probably put you to

bed for some days and tell you to stop work and worry, for a while at least. Then the chances are he will give you a diet that is mostly green vegetables and contains very little meat. If you are fat, he will probably give you a diet that will make you lose weight as well.

While probably no doctor can cure it, high blood-pressure is not a very troublesome thing to live with if found early and looked after properly.

CHAPTER XVII.

BRIGHT'S DISEASE.

If Death were to range his faithful henchmen beside him, Bright's disease would be well toward the place of honor. We have lungs to breathe with, and consumption and pneumonia to attack them. We have a heart to pump the blood about the body, and heart disease to enfeeble it. We have kidneys to keep the blood fit to circulate, and Bright's disease to hinder them. Tucked beside our backbone, just below the last ribs, the kidneys are on the job day and night. They do their work so well that ordinarily we are not aware of their existence.

The task of the kidneys is no easy one. They have to separate from the blood waste material and any excessive amounts of useful material, and at the same time prevent the escape of such substances as the body needs. This alone is a problem that would worry an expert chemist. Besides this, they have to help in maintaining the very exact balance between acids and alkalies in the body, where a variation measurable only by delicate tests would result in death. Finally, the kidneys prevent the blood from becoming too dilute. One of the experiments I remember from my physiology course in medical school was drinking large amounts of water and noting the corresponding increase in the amount of urine.

These different functions of the kidney cannot be performed by a crudely constructed organ. The kidney is made up of a large number of units, emptying into collecting tubules. A single unit consists of a tuft of thin-walled vessels, called a glomerulus, surrounded by a space which leads to a greatly

coiled tube; a long straight loop; and then again a much coiled tube emptying into the collecting tubule. These units are not distinct, but are intimately intertwined. Each unit empties into one of the collecting tubules just as house drains empty into a main sewer. The collecting tubules in turn empty into the expanded end of the ureter, which reaches from the kidney to the bladder.

While theories as to the way in which urine is formed are plentiful, there is one which is fairly generally accepted and harmonizes with the known facts. According to this theory, as the blood passes through the thin-walled tuft, the glomerulus, a part of it filters out into the surrounding space. If everything is working well, the blood cells, the proteins and the sugar of the blood are held back, but some of the water, the waste substances and the salts filter through the walls of the cells making up the tuft. This solution of salts and wastes flows down the tubule. As it trickles along, the cells making up the tubule take up some of the water, so concentrating the urine, and some of the salts, returning them to the blood. On the other hand, they excrete into the urine more waste substances. Thus the final product consists of waste material with such water and salts as the body does not need.

In any machine as delicate as this, there are a good many opportunities for it to get out of order. And since the different parts of a unit are so closely interrelated, serious damage to any one part results in the shut-down of the whole unit. Thus the initial trouble may be located in either the glomerulus or the tubule, but the end-result is much the same in either case.

One of the first evidences of trouble is the presence of albumin in the urine. When the glomerulus has been damaged, it cannot keep back the proteins of the blood perfectly and some of them, shown by chemical tests to be albumin, escape with the wastes in the urine.

There is a considerable variety of conditions, all char-

acterized by the presence of albumin in the urine and lumped together under the name Bright's disease. This name, now going out of date, was used because John Bright, an English physician, was the first to describe the disease. Nephritis is the medical term for damage to the kidney, and is better to use, as Bright's disease includes several separate diseases. Nephritis may be divided into two groups, acute and chronic, according as to whether or not red blood cells are present in the urine.

Acute nephritis is more common in children and young people. The usual cause is the action on the kidney of the poisons of the germs of scarlet fever and of tonsillitis, the streptococci. Exposure to cold and wet is one of the more infrequent causes. Nearly one-third of those stricken with acute nephritis die. Some of the most severe forms, resulting almost invariably in death, are those due to the metallic poisons, such as corrosive sublimate.

The urine in acute nephritis is scanty and dark and contains both red blood cells and albumin. Since the injured kidneys cannot excrete the normal amount of water from the body, it collects in the tissues and dropsy appears. Nausea is common and even convulsions are not rare. Sometimes recovery is complete, but more often, if death does not intervene, the disease passes on to its chronic form.

The treatment of the condition varies with the activity of the disease process. In acute nephritis the aim is to rest the kidneys so far as possible. The activity of the body is cut down to a minimum by rest in bed for at least three months, and the diet is best restricted to milk alone at first, with a little fruit, bread and green vegetables added later, but no meat and little, if any, salt. The amount of fluid taken in must also be kept low, as any large amount piles up in the tissues and adds to the dropsy.

Just how the chronic forms of nephritis are caused is a very much debated question. Some cases can be traced back to a preceding acute nephritis. Others are presumably

due to absorption of poisons from low-grade focal infections, such as abscessed tonsils or teeth. Still others follow hardening of the arteries or high blood-pressure. Some doctors believe that too much protein food may have something to do with the disease.

In the first class of cases, those not related to arteriosclerosis or high blood-pressure, the kidney has trouble in eliminating water as well as wastes, while the albumin, which should be held back, escapes. Before we get too far, it is well to point out that we are talking in generalities, and that there is no hard and fast distinction between the various types of nephritis. This is in some degree due to the unsatisfactory state of our knowledge of the subject.

In the kind of Bright's disease due to hardening of the arteries, or rather which occurs with hardening of the arteries, the outlook is perhaps best. This is the common form in older people. Here it is hard to say what is cause and what is effect. The kidney trouble retains waste products in the blood, makes excretion more difficult, and so causes higher blood-pressure. The high blood-pressure may itself damage the kidneys, or it may cause hardening of the arteries, which in turn deprives the kidney of its necessary blood supply and so causes damage. Lastly, hardening of the arteries may be the first to appear. This may damage the kidney directly, or else do so indirectly, by causing high blood-pressure. All three conditions are as a rule intimately interlocked. Luckily, what is good for one is also good for the others.

High blood-pressure, or at least some increase in pressure, is not an absolute evil. Greater pressure is required to assure the kidneys an adequate supply of blood and to get sufficient excretion through the damaged kidney, just as a greater head is required to force water through a rust-clogged pipe. So even if we had some means of lowering blood-pressure, it is very doubtful whether its use would be advisable in cases of Bright's disease.

Strangely enough, in spite of all the work done on nephritis and our elaborate methods of analyzing the blood, we do not as yet know the substance that causes the symptoms of uremia and convulsions. This substance is probably in some way related to the nitrogenous wastes, because symptoms increase as they pile up in the body, yet extended chemical studies have shown that they themselves are not at fault. They are an index of the amount of kidney damage, and an indication of when trouble may be expected, but that is about all. Perhaps some day the trouble-maker will be found and our efforts at relief will be more successful.

In the form of Bright's disease associated with hardening of the arteries the outlook for the sufferer is the best. The albumin in the urine is a warning signal to ease up a little. We do not junk an automobile if it begins to knock on the upgrade, but shift gears.

It is just as well to recognize that Bright's disease is incurable. The changes in the kidney can no more be reversed and the injured tissues restored than hair can be grown on a bald head. But because incurable it is not hopeless. There is no reason why a person who has some hardening of the arteries, somewhat increased blood-pressure, and some albumin in his urine cannot live a happy and useful life.

The treatment of chronic nephritis can be summed up under three general headings: prevention of further damage to the kidneys; prevention or removal of dropsy, and an adequate supply of food. The first thing to do is to see that no more injury occurs. Foci of infection, such as bad teeth and tonsils, should be cleared up. Care should be taken to avoid absorption of chemical poisons, such as mercury or lead compounds. The diet should be kept low in proteins, as the end-products of proteins are the hardest to get rid of. The diet also should not contain too much acid. The total intake of food ought to be rather low, so that the kidneys will have a minimun amount of wastes to excrete. Finally, mental and physical rest is valuable.

Dropsy can be taken care of in several ways. The amount of fluid and of salt taken in must be kept fairly low. The fluid already dammed up in the tissues can usually be driven out by sweating and cathartics.

Maintaining an adequate supply of food is not easy. The fuel value should be such as to keep a slightly underweight condition. The proteins and fluid must be determined largely by the ability of the individual to take care of them. The foods should be chosen so as to include a sufficient supply of lime, iron, and other minerals, and also of vitamins.

With these principles of treatment applied, chronic Bright's disease should lose some of its unsavory reputation, though it will probably always rank as one of the "Captains of the Men of Death."

CHAPTER XVIII.

RICKETS.

The sacred codfish that hangs in the Massachusetts State House won its place by virtue of its flesh. Important as is the cod in the fish markets of today, from the doctor's standpoint the cod-liver oil is worth far more. Cod-liver oil is a substitute for sunshine.

Lack of sunshine causes rickets. Babies born in the fall and early winter are the most apt to develop rickets, for through the winter and early spring they are kept indoors, away from the sun and air. Dark-skinned babies, such as those of the negroes and Italians, are peculiarly susceptible to rickets, for the pigment in their skin still farther reduces the amount of ultra-violet light which can penetrate into their bodies.

From fall until April or May increasing numbers of patients will be brought into the children's wards of our hospitals. Some babies will be restless, constipated, with sweating of their heads. Others will be more seriously ill, softening of the bones, often with fractures, bowing and severe deformities. In some, the bones of the skull are so far thinned that they bend and crackle like heavy wrapping paper when pressed by the fingers. The joints, or rather the bones near the joints, are tender and swollen.

Many of the deformities in later life are due to the damage done by rickets in infancy. Inasmuch as the injury is to bones, its effects last not only during the period of active disease, but throughout life. The very severe cases of bow-legs, many deformities of the chest, and many of the structural changes in the bones of the pelvis which make child-

birth extremely dangerous for some women are among the troubles that this disease causes. The soft bones of the baby need lime salts to stiffen them and fit them for weight-bearing and resistance to the pull of muscles. Rickets hinders this deposit of lime and at the same time stimulates to active growth the soft tissue which is the foundation of bone.

At bottom the disease is due to an inability of the body to store and lay down in the bones lime and phosphorus. Mere feeding of lime and phosphorus is not enough. The baby with rickets may be getting all the lime he needs in his food, but the trouble lies deeper than merely the available supply. The lime is not absorbed from his digestive tract, or if absorbed is not used, and does him no more good than a bath in lime water.

Technically speaking, there are two forms of rickets, one in which the available lime in the blood is low, in the other the phosphorus. But fundamentally these are the same—same cause, same symptoms, same cure. The difference between the two can be told only by elaborate chemical tests. We can forget about it without being any the worse.

In the opinion of the leading specialists in children's diseases, today rickets is almost universal among the infants of the poor in cities, and it is by no means restricted to the tenement dwellers. In children from three months to two years old it is probably the most common of all diseases.

The seasonal distribution of the disease is striking. Twice as many cases come to the hospitals between January and June as during the rest of the year. From the evidence of postmortem examinations, the early cases of the disease practically all appear in late winter and spring, while in the summer and fall the cases show evidence of healing.

Though the disease has been known since 1650, only the last few years have we learned much about its cause. The first hint came from a study of the occurrence of rickets. Here is a condition very common in the cities of Europe and North America, particularly the industrial centers, yet

very rare in the country. It is common in domesticated animals, and wild animals kept in captivity, as in zoos and circuses, but never is found in wild animals in their native state. Curiously enough, the cat, which man has never completely tamed, and which still finds "wild, wet woods," even in city alleys, never develops rickets.

Evidently the disease has something to do with the conditions of civilized life. Yet, as is the case with most obscure diseases, it has at one time or another been blamed on many things.

Thus some workers have tried to prove that it is hereditary, others, including Koch, the discoverer of the tuberculosis germ, have blamed it on one or another of the bacteria, and many have claimed the trouble lay with one or another of the glands of internal secretion. One of the favorite theories has been that the cause was a deficiency in the secretion of the thymus gland, a mass of soft tissue lying in the upper part of the chest, which does not seem to have any particular job. The thymus is most highly developed in infants, and gradually disappears until at the end of the growth period only a trace of it is left. The theory was a nice one, but it was not true.

With the knowledge of the importance of diet in relation to health and disease, and especially of the need for an adequate supply of vitamins, the effect of various foods on rickets was studied. Five years ago, Dr. Mellanby, an English research worker, reported the results of feeding various diets to puppies.

He found the more bread the diets contained, the more severe cases of rickets his puppies developed. Practically all the puppies got rickets unless they were fed either cod-liver oil, butter fat, suet, peanut oil or cocoanut oil. Feeding of cod-liver oil always prevented rickets, the other oils and the fats were not always satisfactory.

The better balanced his diets were, the less cod-liver oil was needed to prevent rickets. The older the puppy, the

poorer was the diet that could be eaten without causing the disease.

In other words, Mellanby and the other workers who followed his lead showed that rickets could be caused by the absence of an "antirachitic vitamin" from the food and that the disease could be cured or prevented by feeding cod-liver oil, which is rich in that vitamin.

But this was not all. Rickets may develop on almost any food combination or milk mixtures ordinarily given to children. We have already spoken of the prevalence of the disease in the cities and its tendency to increase in late winter and spring. Moreover, cases are most severe where overcrowding and poor hygiene are the rule.

These facts gave another lead. When rickety children were taken from dark tenements and put in open sunlight, they got better. Country babies, much in the sun and open air, almost never get rickets.

The most active part of the sun's light is the invisible or "ultra-violet" portion. This part of the light is filtered out by window glass, and there is much less of it able to get through the atmosphere in winter than in summer. Also, pigment in the skin partly insulates, so to speak, from the sun's rays, so that negro or dark-skinned children need more sunlight than those with fair complexions. It had long been known that negro and Italian children were specially susceptible to rickets.

The facts began to fit together like the pieces in a solved picture puzzle. When actual experiments were done, it was found that direct sunlight, or artificial ultra-violet light from quartz lamps prevented the disease and cured it when present.

So it seems that today we have the answer to the puzzle. Rickets is a deficiency disease, caused by the lack of an "antirachitic vitamin" in the diet, or by the absence of ultra-violet light in the environment, or by both. It can be cured by cod-liver oil or by ultra-violet light or sunlight in the open air. It is about the only known disease with two causes and

two cures. Perhaps some day it may be divided into two distinct types.

This two-fold nature is well shown by the African and Esquimaux babies. The African babies live out of doors in the sunlight, and rickets is unknown. The Esquimaux babies live in dark huts, the sun shines only one-half the year, and rickets is unknown. The African baby gets sunlight—the Esquimaux gets fats rich in antirachitic vitamins—and both escape rickets.

In cities sunshine is none too plentiful in summer and rare in winter. What there is has had most of its valuable ultra-violet rays filtered out by the dust and smoke in the air. We have an expensive substitute sunshine in the ultra-violet light given off by a mercury arc in a quartz tube. Some hours of exposure to ultra-violet light, spread over many days, will prevent or cure rickets.

A better substitute for sunshine is cod-liver oil. It is not used as the whale oil the fleets of Nantucket and New Bedford brought us in the forties; not as a giver of light, but a giver of life and health.

For centuries the fishermen of Holland have given their children cod-liver oil; their children have no rickets. Cod-liver oil is an old remedy in New England as well, though most of us made its acquaintance through the desire of our mothers to ward off colds and fatten us up. Only in recent years has it come into its own as the outstanding remedy for rickets.

Cod-liver oil to be effective does not have to be nasty. There are two varieties. One is the old-fashioned dark brown kind, to make which the livers were left to decompose and then the oil skimmed off. In the process generally used today the oil is melted out of fresh livers by steam. This oil is pale, and with little smell or taste.

Recently Dr. Steenbock of the University of Wisconsin made a remarkable discovery which throws a little light on the nature of the antirachitic vitamin. He was carrying on some experiments on rickets in animals, and found that when

a diet deficient in antirachitic vitamin was fed to animals treated with ultra-violet light, they did not develop rickets. Animals on the same diet, but not radiated with ultra-violet light, soon showed the characteristic changes of rickets. This was all entirely fitting and proper. But when he fed his animals just the same diet, except that the food had been exposed to ultra-violet rays, they stayed perfectly healthy. In other words, by treating the fats in the food with ultra-violet light he supplied it with the antirachitic vitamin which was missing. So even if there may be a scarcity of cod-fish, we can still have our supply of antirachitic vitamin.

With sunlight, ultra-violet light, and cod-liver oil at our disposal there is little excuse for rickets to exist today.

CHAPTER XIX.

HAY FEVER.

The colder and longer drawn out the spring is, the more comfort and relief some people have. No, they are not furriers or raincoat manufacturers. They are just like you and me, except—and this a big exception—they have hay fever. Flowers may look and smell just as good to them as to the rest of us, but sneezing, sore eyes, running nose and asthma make up the price they have to pay for enjoying them.

As in the case of a number of the annoying but not serious diseases, most of the pioneer work in trying to find the cause and develop a rational method of treatment has been done by physicians who themselves were suffering from hay fever. The first careful study of the disease was made by an English doctor, John Bostock, a little over a hundred years ago, though he had no suspicion of the cause. Most people at that time called it hay fever, though the doctors preferred to blame it on hot, poorly ventilated houses, and sometimes on the odor of various flowers.

About seventy years ago another English physician, Dr. Blackley, got tired of passively sneezing and wheezing through the summers. He noticed that his symptoms were worse on hot, dry, windy days, when much pollen might well be in the air. But how was he to prove that pollens were at fault? He collected the pollen from the flowers of over a hundred plants and then proceeded in the true scientific spirit to experiment on himself. He took samples of the different pollens, inhaled them, rubbed them into his eyes, scratched his skin and rubbed them into the scratches. Sure enough, he developed hay fever each time, but the

symptoms were much worse when the pollens of grasses were used.

So far, so good. He next exposed to the air glass plates covered with sticky substances and examined with a microscope the material that had been caught on them. On the days his hay fever was bad, the glass plates revealed lots of pollen grains. When only a few grains were caught, his symptoms let up. On careful examination of the pollen grains, he found that nearly all of them were from flowering grasses. (Ragweed sufferers please note: this was in England.)

What is there about an innocent seeming substance like the pollen of a plant that makes it cause so much trouble? Pollen contains protein, all plant and animal cells do, and each species of plant and animal has its own protein which is just a little different from every other. They may all be made on the same general principle, just as the locks of safes, but each has a different combination.

We all know people who get hives after eating lobster—I do myself after eating shrimps. In their case it is the effect of the lobster protein on their bodies. When a protein and a person sensitive to that protein meet, trouble is in sight. But the same protein will have no more effect on a normal person than the wrong key has on a lock. It is natural that related plants should have nearly the same proteins in their pollens, which explains the common observation that people sensitive to timothy pollen, for example, get disagreeable though less marked symptoms when they come in contact with pollen from red-top.

But curiously enough, this relationship does not extend to different parts of the same plant. A man who gets hay fever from timothy grass pollen ordinarily shows no effect at all on contact with even large amounts of the stalk or blades of the grass. To make a rather crude analogy, a hen's egg is more like a duck's egg than it is like the hen's drumstick.

Our bodies are pretty delicate machines. They have their own proteins, and when any other strange protein gets in, it makes trouble. We eat large amounts of foreign proteins, such as are in meat, cheese, eggs, or nuts, but the digestive juices break them down to simple compounds called amino-acids which can be harmlessly absorbed by the body and then either be used for production of energy or be recombined to form the various proteins found in humans. The general opinion is that when a person is "sensitive" to a certain protein, in some way that protein has gotten into the body tissues unchanged. Perhaps the mucous membranes of that person have not been a strong enough barrier and some of the protein has gotten through without being broken down to its constituent amino-acids. As a result, the person is "sensitized." After that, the least contact with that particular protein makes trouble.

Say ragweed pollen protein has in some way worked into the tissues of one's body. After that, if one breathes in ragweed pollen, the lining of the nose swells, with all the symptoms of a cold in the head. If the pollen gets further down, to the bronchial tubes, the same thing happens, and in addition the muscles in the walls of the finer air passages contract causing asthma. If it blows into the eyes, they smart, redden, and the tears flow. If it gets on a scratch, redness, swelling and itching develop. In other words, it causes all the symptoms that drove Dr. Blackley to do his experiments on pollens and hay fever seventy years ago.

There is sometimes a considerable mental element in the disease. Instead of the protein, the mere sight or smell of the plant which produces it may be enough to bring on an attack. I remember a woman sensitive to rose pollen who developed a typical hay fever attack when she was shown a rose made of silk.

To treat a case of hay fever, the first thing to do is to find out which pollen is at fault. The range can be narrowed

somewhat by knowing what pollens are in the air at the time the disease comes on. Then extracts in water or dilute salt solution containing the proteins from the various suspected pollens are rubbed into scratches on the patient's arm. After a few minutes redness, itching and swelling will appear about the scratches on which the offending protein has been rubbed. The other scratches will show nothing, or only a very slight change. Instead of scratches, injections of small amounts of the extract into the skin, similar to those used in the Schick test, may be employed.

Once the offending pollen has been detected, the hay fever victim can be freed from his trouble, or "desensitized" by injections of very minute amounts of the trouble-making protein. Occasionally the treatment will not bring relief, but the cases of disappointment are relatively few. In the greater part of the United States we can recognize three main hay fever seasons. The first begins with the blossoming of the willows and birches in April, and ends early in June when the pines are in bloom. So any one that gets hay fever at this time can safely blame it on the trees. Not many people, however, suffer from this early spring type, and then only for a few days. Therefore, generally speaking, these people do not need to be desensitized. Of course the hay fever may be considerably stretched out if one is unlucky enough to be sensitive to several trees that pollinate one after the other, such as willow, birch, oak, alder and pine.

The second season begins in June, when the Kentucky blue grass blossoms, but really hits its stride toward the end of June and lasts into late July with the red-top and timothy season. Occasionally the daisy and plantain pollens make a little trouble, but this is the exception rather than the rule.

The third season is our busiest one. About the middle of August in the northern part of the country, and earlier the further south you go, the ragweed begins to bloom. Simultaneously the big hay fever season begins, and lasts up to the killing frosts of late September. We have all seen road-

side ragweed patches that gave out clouds of yellow pollen when disturbed. The pollen is so light and so abundant that it penetrates everywhere. Even the down-town sections of large cities are not safe for the ragweed sensitive person. The plant itself is nearly as widely distributed as its pollen. Vacant lots, gardens, railroad yards all have their quota. Not content with making itself obnoxious, the ragweed gives other plants a bad name. It scatters its pollen over golden-rod and wild aster, so when anyone passes by the ragweed pollen reaches him, though the trouble appears to be caused by the golden-rod or asters themselves. The one relief Nature provides for the ragweed victim is heavy rain—this wets down the pollen and clears the air for awhile. The only other escape, aside from finding a country where ragweed does not grow, is the desensitization treatment.

Occasionally other proteins than plant pollens make trouble. Some of these are very amusing, though rare. One of the queerest I have heard of was the case of a young man who got hay fever from the rice powder his sweetheart used. Luckily she was persuaded to change to talcum powder and the story has a happy ending. Some people are sensitive to the hair of cats or dogs. A classmate of mine in medical school had violent hay fever whenever he got near a horse. But these scattered cases are dwarfed by the vast number of cases of hay fever due to the plant pollens.

CHAPTER XX.

PTOMAINES AND FOOD POISONING.

Ptomaine poisoning is a term we use a good deal, without bothering very much about the exact meaning of it. If we eat anything that disagrees with us, particularly if we have been to a banquet, we blame it upon ptomaines. But as to what they are, we have only a vague idea.

When the bacteria of putrefaction act on proteins such as meat or fish or cheese, some of the protein is broken down to organic chemical compounds, varied in nature, which are mildly alkaline. These are called ptomaines. They are not all harmful, in fact, most are not. As the meat or cheese decomposes, the poisonous ptomaines are formed gradually and reach their height soon after the seventh day, as a rule. From then on, they decrease in amount, and finally disappear entirely, being broken down to more simple and harmless chemical compounds. Whether the same thing happens in the case of fats is not definitely known, but there are several substances that are highly poisonous which may be formed by the break-down of fats in the laboratory.

Most of the poisonous ptomaines are dangerous only when injected directly into the blood; when swallowed they are, with a few exceptions, not very harmful, owing to the destructive action of the digestive juices on them or the slow absorption from the digestive tract which permits the liver to reduce or entirely remove their poisonous characteristics.

The ptomaines differ from the specific toxins formed by some bacteria, such as the diphtheria or the botulinus bacillus, in that they are due to the breaking-down of the proteins on which the bacteria are growing while the toxins

are built up synthetically. Thus no matter on what protein the diphtheria bacillus grows, its toxin is exactly the same, while the ptomaines formed differ widely with the various proteins.

Curiously enough, ptomaines can be produced by many germs which are incapable of causing any disease. Some of these poisonous products are very similar chemically to the active principles of poisonous plants, such as deadly nightshade or the Amanita toad-stool.

It is safe to say that if there are ptomaines in food, germs are also present unless it has been sterilized. If the germs are harmful ones, the damage they can do far out-weighs that of any ptomaines that may be present. The commonest harmful bacteria present in food are the members of the so-called meat-poisoning group. They are not unlike the bacillus of typhoid fever in many respects, and produce symptoms similar to typhoid, though less severe. When numbers of these germs are swallowed we have what is commonly called ptomaine poisoning, coming as a rule eighteen hours or more after the spoiled food has been eaten. The attack is long or short, severe or mild, according to the particular kind of germ and the numbers of them that were present in the food. In other words, the illness is due to the bacteria themselves and not to the ptomaines they may have formed.

There are many varieties of these germs, but their action is about the same, whether the bacteriologist labels them Bacillus enteritidis, Bacillus paratyphosus B, or Bacillus suipestifer. The vast majority of outbreaks of food poisoning are due to one or another of these germs. Banquets afford a special risk, because of the favorable conditions of growth offered the bacteria, where large quantities of food are prepared before-hand and kept warm for some time. Moreover, the large amount of handling that takes place in the preparation of the food gives an excellent chance for germs to get into it. Such dishes as salads or macaroni are particu-

larly apt to be dangerous, the first due to the large amount of handling necessary, the second because it is cooked in large pans so that the center is hardly more than warmed, really providing any bacteria there with ideal growth conditions. But after all, the number of food infections is very slight considering the number of meals we eat.

Besides these bacteria of the food-poisoning group, the germs of typhoid fever may occasionally make trouble, though this is now a rare disease, thanks to the vigilance of our physicians and health officers.

One of the great blessings of modern life, but one readily abused, is cold storage. Cold is an excellent preservative of food. Just how long meat can be kept is shown by the discovery of the fairly well preserved flesh of mammoths, frozen thousands of years ago, in Northern Siberia. But when cold storage is used to "corner" a commodity, or raise prices artificially, the temptation is to keep the food in storage longer than it should be kept. Many state departments of public health set a limit of one year on the time which foods may be preserved in cold storage before being sold. Occasionally an extension of time is allowed if the food is in good condition. The departments also generally require that any store selling cold storage goods display a sign to that effect.

One peculiarity of cold storage food is its rapidity of decomposition, once removed from the cold. This means that such food is liable to produce poisonous ptomaines in a shorter time than fresh and affords a better place for germs to grow.

The rapidity of spoilage of cold storage chicken is particularly noticeable. Not long ago I had a mild attack of ptomaine poisoning that could be traced directly to chicken. A number of other doctors who had eaten the same meal shared the trouble with me. In this case the upset was probably due to a ptomaine rather than the bacteria, as the symptoms came on in a very few hours and soon subsided. This matter of time between eating the food and appearance

of symptoms is an important one for diagnosis of the condition. In true ptomaine poisoning trouble shows itself in a very short time, rarely over eight hours, after eating the spoiled food. The poisons are already formed and begin to act just as soon as they are absorbed. On the other hand, food poisoning due to germs themselves does not develop until the germs have gained a good foothold and have multiplied to a considerable degree. Therefore in this type twelve or eighteen hours or longer elapses before much of any trouble is noticed.

The rapid decomposition of cold storage chicken is largely due to the spreading of bacteria throughout the flesh. If the poultry has been drawn in the ordinary way, the intestines break below the gizzard and allow contamination of the carcass. Usually fowl are not drawn, because of the trouble saved and the profit to the dealer from the weight of the entrails. This also permits spreading of germs from the intestinal tract throughout the body.

The ideal way is to store fresh-killed fowl which have been carefully drawn, removing crop, gizzard and intestines in one mass. If this were done, the danger of trouble from cold storage chicken would be very slight indeed.

Vegetables ordinarily are not thought of as a source of disease, but the occurrence of small epidemics of botulism from eating canned spinach, ripe olives, and other canned vegetables has taught us that they must be considered. However, the toxin secreted by the Bacillus botulinus, the cause of botulism, does not give the usual digestive upset we associate with food poisoning. Instead, the nerves are attacked. Symptoms develop generally sixteen to thirty-six hours after the food containing the toxin has been eaten. Fatigue, dizziness and apprehension are followed by dimness of vision and seeing double. Then difficulty in speaking and swallowing develops and attacks of strangling come on.

Luckily the methods of preserving food used at present practically rule out any occurrence of the toxin in commer-

cial canned goods. Ordinary care in thoroughly heating food before serving, simmering for ten or twelve minutes for example, will absolutely destroy any toxin that may be present.

In any consideration of food poisoning, the natural poisons, such as poisonous mushrooms, should have a place, even though small. They are usually met with in children who do not know enough to avoid the poisonous forms. Occasionally a naturally good food may become poisonous. Thus the potatoes which have been turned green by exposure to the sun contain a poison called solanin. Rye blighted by the fungus or ergot may cause either acute or chronic ergot poisoning, the St. Anthony's fire of the Middle Ages.

Bacteria and plants are not the only ones that can throw our digestive system out of order. The embryos of a parasitic worm, trichina spiralis, are the cause of severe and frequently fatal disease. The embryos are present in hogs, sometimes so plentifully as to give the pork a "measly" appearance, and are not killed unless the meat is thoroughly cooked. When living embryos present in the pork are swallowed the meat surrounding them is digested and they are set free. The embryos pass into the small intestine and develop there during about three days. Then after three or four days more each adult worm sets free several hundred embryos which make their way into the blood stream and invade the muscles.

If only a few embryos are eaten, no symptoms at all may result. The disease as a rule shows itself within one or two weeks after the infected meat has been eaten, appearing as fever, then pain in the muscles and swelling of the face. Recently several small outbreaks, each involving a single family, have occurred in Boston. Several of the persons infected died. Here again all danger is removed by thorough cooking of the meat.

All these things I have spoken of so far affect one person as well as another. A few may have only a slight upset

while others are made seriously ill, according to the amount eaten and the general condition of the person eating it. But there is much truth to the old saying, "One man's meat is another man's poison." We all know people who are made deathly sick by lobster or scallops or veal—the list of foods can be stretched out indefinitely—while others are unharmed.

Here the trouble does not lie with the food but with the person eating it. The person has become what is technically known as "sensitized" to that particular food in some way, and eating it will invariably cause trouble. This form of intestinal disorder can be compared to hay fever. In hay fever a person may react to a particular pollen when people all around are breathing in the same material without the least difficulty. The victim of hay fever is sensitive to the protein of his particular *bête noire* among the plant pollens, just as the victim of food sensitiveness is upset by the protein of the food he is susceptible to.

For the sufferers from this type of food poisoning there is practically nothing to do. They must learn to avoid their particular poison.

There is really little excuse, however, for the ordinary type of "ptomaine" or food poisoning. Good food and thorough cooking, with ordinary care in handling, would practically wipe out all the forms I have mentioned, except the last, in which the person and not the food is to blame.

CHAPTER XXI.

AUTOINTOXICATION.

The heading of this chapter does not mean that I am going to write about drunken driving, however much it may sound like it. The word autointoxication comes from two Greek words, meaning self and poison. Intestinal autointoxication is the injury done the body by poisons formed in and absorbed from the intestine.

We know that when we are constipated we can be very uncomfortable, and that a thorough cleaning out gives prompt relief from such unpleasant symptoms as nausea, headache and general discomfort. We know that the contents of the lower bowel may be potentially poisonous. Here then is a plausible explanation of our ills. Absorption of poisons from the retained intestinal contents, according to the enthusiastic advocates of the autointoxication theory, causes headache, nausea, high blood-pressure, kidney trouble, cancer, even sudden death.

Not entirely disinterested in their crusade to make us well by prevention of autointoxication are the patent medicine men whose advertisements of laxatives confront us from bill-boards, street cars and drugstore windows. Mind, I hold no brief for constipation. Its power to do harm is beyond question. But there is some doubt as to just how much of the harm blamed on autointoxication really belongs there.

The possible sources of poisonous substances which may be formed in the intestine are three. The first and least important is the bile and digestive juices. These so far as we know cause no harm if absorbed into the body, though

a few experiments in which fairly large quantities were injected directly into the blood stream have shown they have some potentially dangerous properties. The bile may act on the lining of the intestine to a slight extent so that poisonous substances or bacteria may pass through more readily than is normal.

The second source is the food, either the proteins or the fats. Under normal conditions the digestion of protein results in a large number of substances, some of which may be injurious. The proteins are changed to amino-acids by the digestive juices. If some of the intermediate compounds known as proteoses and peptones, gain entrance to the blood stream, severe trouble results. However, these compounds cannot pass through the wall of the intestine unless it is injured and so made a less efficient barrier. In a consideration of autointoxication these difficultly absorbed compounds may be omitted.

The third possible source of poisons is the action of bacteria on food. When certain forms of bacteria are present in large numbers, the amount of harmful substances may be very considerably increased. In this group the toxins formed by certain germs, such as the dysentery bacilli, may also be included.

Metchnikoff, for many years in charge of the Pasteur Institute, believed that the decomposition products of protein due to the action of putrefactive bacteria present in the bowel were the chief cause of the ills of autointoxication. His book, "The Prolongation of Life," had a wide circulation, and did much to popularize the autointoxication theory. He believed that the protein bacteria could be driven out by a germ which lived on carbohydrates, the decomposition products of which are practically harmless. Bulgarian buttermilk or koumys contained this germ, and drinking the buttermilk was supposed to give the germ a chance to establish itself in the intestines, driving out the harmful forms.

Unfortunately, careful study has shown that the Bacillus bulgaricus, to give it its full title, cannot hold its own against the protein destroying germs. However, we have now available a related germ, the Bacillus acidophilus, which does very well. The advantage of having such a germ within us instead of ones capable of forming poisons from the protein of our food is obvious.

Before we get too far, it may be helpful to review the functions of the different parts of the digestive tract. The mouth prepares the food to be swallowed and the esophagus carries it to the stomach, which starts the process of digestion, especially for protein foods. The stomach does not absorb any of the food, although there is some evidence a few drugs are taken up here. In the uppermost part of the small intestines, the duodenum, there is very little absorption, more digestive juices are added to the food, and small amounts of certain body wastes are excreted. The remainder of the small intestine absorbs the digested food. When the first part of the large intestine is reached, a small portion of the digested food is taken up and absorption of water from the residue begins. Through the rest of the large intestine a large amount of water is taken up and small amounts of waste are excreted, but the absorption of substances other than water is negligible. The last portion of the large intestine is little more than a storage place for feces. Thus from what we know of physiology, the evidence is rather against the absorption of poisons from the intestinal contents in that part of the bowel which is filled up in constipation.

The symptoms of constipation are so familiar that it hardly seems worth while to name them over: tiredness, headache, biliousness, coated tongue and loss of appetite. We have all had them and we all know that they vanish practically at once after a bowel movement. This is perhaps the strongest point against absorption of poison substances as a cause for the symptoms. Even if all the poison in the intestine were removed by the bowel movement, the effects

could not disappear so rapidly, as that already absorbed could not be thus removed. A sponge does not become dry immediately after being taken out of the water.

Under carefully controlled experiments in the laboratory it has been shown that the dulness of constipation is a very real thing, not merely a feeling, and the almost instantaneous relief felt from a bowel movement is equally real. The ability to do muscular work, the chemical constitution of the blood and the mental activity all show definite changes.

Plugging of the lower end of the digestive tract slows down, stops, or reverses the waves of muscular contraction which carry the food along the intestines. As the food cannot go downward because of the plugging, the bowel tries to force it back toward the mouth. This attempt of the intestines to avoid over-loading results in the belching, coated tongue, and bad breath. Removal of the plug relieves all these.

This is a very plausible explanation, but it is not enough. There are a number of people suffering from such symptoms who do not experience relief from a bowel movement. For these cases, a definite chemical autointoxication must be assumed.

We know that in children, and particularly infants, the lining of the intestine is very delicate, and poisonous substances can readily be absorbed from the intestinal contents. The products of bacterial action in dysenteries and diarrheas in children are often very poisonous and cause severe symptoms when absorbed.

Recent research on the mode of action of the germ of Asiatic cholera, probably the most fatal of all intestinal diseases, has shown that the cholera germs attack and kill the lining of the upper intestine, and that the absorption of this decomposed intestinal lining by the intestines lower down causes most of the trouble. In other words the symptoms of the cholera patient are due to his own body

proteins changed into poisons by the action of the cholera bacilli.

Dr. Kendall in Chicago has shown that even some of those germs which produce definite poisons may form none or very little if given a plentiful supply of starches or sugars, but when only proteins are available the poison production is at its peak.

The absorption in the case of the cholera poisons occurs chiefly in the lower parts of the small intestine, and it is likely that in the group of cases which may be classed as true autointoxications the poisonous substances in the intestinal contents work back from the large intestine into the lower end of the small. There is a fold of mucous membrane, the ileo-colic valve, which has the duty of keeping the material in the large bowel from working backward into the small. However, in a digestive system upset by constipation accurate functioning of this valve would probably not be possible.

Whatever the reason may be, there is a great deal of evidence which goes to show that a number of chronic disease conditions are associated with constipation. If, as seems likely there is a little absorption of poisons from the intestinal contents in most of the people suffering from what we commonly call autointoxication, what is the trouble? In most of these the obstruction of the normal passage of food through the bowel or attempts to force it back explain the symptoms. Yet that leaves unexplained the chronic diseases, such as rheumatism and nervous disorders, so often occurring in constipated people.

We have for a long time assumed that germs get into the body tissues only in grave disease, blood poisoning, typhoid fever, pneumonia— and that normally the various tissues are sterile. However, under certain circumstances germs may pass into the body through the intestinal wall. In all probability, constipation helps to bring about favorable conditions for this passage.

Most of the germs that get in are rapidly destroyed in the blood or lymph nodes, without ever having gotten very far. A few may secure foothold for a greater or less time in various parts of the body, especially where the tissues are in some way damaged and have low resistance. The presence of these germs, or the strain of having to kill and dispose of them repeatedly, may result in slight but gradually cumulative damage, until a full-blown disease appears.

One more possibility is that certain substances, such as the "bios" that stimulates tremendously the growth of yeast, may occasionally get into the bowel and cause normally harmless bacteria to produce very poisonous substances.

The question is far from settled, but however it comes out, we know that daily bowel movements are normal for most individuals and we feel badly without them.

CHAPTER XXII.

APPENDICITIS.

Common as it is today, forty years ago the word appendicitis was unknown and the disease it describes was called "inflammation of the bowel" or "perityphlitis" and was almost always fatal. To operate for relief of the condition was practically unheard of. Ever since Dr. Fitz of Boston in 1886 showed that the vermiform appendix was the location of the trouble our methods of treatment have improved, as well as our methods of diagnosis. Thus in 1895–1896, 82 cases were operated on at the Boston City Hospital for appendicitis and over one-fifth of them died, due in most part to the advanced character of the disease. In 1921–1922 at the same hospital, only 3 per cent of the 913 cases operated on died, and the ones that died were brought to the hospital so late that they had hardly a fighting chance.

The appendix is a good deal like the tonsils in a number of ways. We are not quite sure what it is good for; we seem to get along just as well without it as without tonsils. It appears to be the weak point of the intestinal tract, the one most easily invaded by bacteria, as the tonsil is the weak part of the throat. The analogy between the two organs is really very striking. The appendix is very rich in lymphoid tissue and the tonsil is little more than a mass of the same material. Just as various germs can cause trouble in the tonsils, so appendicitis is due to a number of different organisms, and in each case the germ called streptococcus is one of the chief offenders.

The appendix is not a very startling organ to look at. It is just a shade smaller than one's little finger, as a rule, though I have seen some as long as a lead pencil. The

small intestine does not connect with the large bowel end-to-end, but enters it at the side, 1 or 2 inches up from the end. At the tip of the resulting blind end of the large intestine is the appendix. If we could project its position on the skin, it would usually lie on the right side, about 1 inch above and 2 inches toward the center from the hip bone. It is a hollow, thick-walled tube, with one end blind and the other entering the large intestine.

In some animals the appendix is far more important than in man. The porcupine glories in one a foot long and three or four times the diameter of that in the human. As a rule, the lower the nutritive value of the food an animal eats, the more prominent the appendix is.

If a cross-section of the appendix is examined with the microscope it shows several layers in the wall. Immediately surrounding the central cavity are numerous small glands which secrete a slimy mucus to keep the contents of the appendix moist and protect the walls from injury. Outside these are masses of lymphoid tissue very similar to those found in the tonsils and adenoids. Then comes a layer of connective tissue which surrounds and supports the lymphoid masses. Next there is a layer of smooth muscle fibers arranged circularly and just outside this another layer with the fibers running longitudinally. Covering all is the peritoneum, the lining membrane of the abdominal cavity.

What the appendix is supposed to do, we do not know. We called it a "residual organ" and let it go at that. Presumably it once had an important part in digestion. The cavity within it contains food material and many bacteria. Normally these are kept churned up and are driven out from time to time by contractions of the smooth muscle in the outer coat. When all is going smoothly we have no sensations from the appendix. But when trouble comes, we usually know it only too well.

There are always germs present within the cavity of the appendix, germs which have found their way in from the

large intestine. These germs practically all produce in greater or less amounts substances injurious to the tissues. So long as they are kept moving, and their injurious products are driven out, no harm results. But if in any way the outlet is blocked or the muscles do not stir things up sufficiently, the poisons accumulate and make trouble.

A fairly wide-spread notion is the one that grape seeds or date seeds cause appendicitis. This is due to the finding of hard masses shaped like seeds in appendices removed at operation. On close examinations these masses are usually found to be hard lumps called fecoliths, made up of bits of food matted together, dried, and sometimes made stony hard by the presence of lime salts. If one of these fecoliths blocks the cavity of the appendix, the germs in the cut-off portion have a fine chance to let their poisons accumulate. A kink in the appendix, shutting off a portion just as a kink in a tube shuts it off, has the same effect.

Even without obstruction, if there is injury to the smooth muscle layers, whether scarring from previous attacks or merely temporary paralysis from one cause or another, so that the muscle does not keep the contents on the move, the same opportunity for poison production is given.

If we could follow through a typical attack of appendicitis right on the battleground, so to speak, we would get a picture about like this. First the accumulated poison kills some of the gland cells and the lining cells. This gives the germs themselves a chance to enter the tissues and multiply, forming more poison. In response to this invasion, pus cells appear and try to overcome the invading organisms. The infection spreads through the lymphoid tissue into the muscle, and the wall becomes filled with pus cells. This increased number of pus cells means more than normal have to be present in the blood in order to supply enough to the fighting line. One of the important signs in the diagnosis of appendicitis is finding an increased number of white cells in the blood.

From this stage two possibilities are open. The defenses of the body can get the upper hand, or the germs will win out, working their way through the appendix and causing it to burst. Once the appendix has ruptured, or even before, the germs can infect the abdominal cavity and set up a peritonitis. Since we have no way of telling what the outcome may be, and only too often the germs do break through, the only safe way is to have the appendix removed just as soon as the diagnosis of acute appendicitis has been made.

However, let us suppose the body defenses took care of the infection. Then as healing begins, the pus cells are replaced by somewhat similar cells called lymphocytes, and scar tissue replaces the killed muscle fibers. If any of the gland cells are left, they multiply and form new glands, and eventually the appendix looks much as it did before, with only scars in the muscle and scattered masses of lymphocytes to bear witness to the fray.

Sometimes all the gland cells are killed off, and in that case the glands cannot reform and the entire cavity of the appendix fills up with scar tissue. In such appendices as these, where there is of course no chance for germs to get in, there is no danger of further trouble.

Strictly speaking, there is no such thing as chronic appendicitis. What is commonly called this is really a series of slight attacks of acute appendicitis, each one leaving a little more scar tissue in the appendix, and so tending to more attacks and eventually to obliteration with cessation of trouble. Whether it is necessary to operate on people who have appendices subject to these recurrent attacks is an open question. Usually they will take care of themselves, but occasionally operation may be necessary. For any one living in or near the city where prompt medical attention can be obtained, operation in this type of case is hardly worth while. But if a trip to the Arctic or elsewhere beyond the rim of civilization is planned, the appendix should be

taken out lest it make serious trouble when there is no help at hand.

While very rarely serious cases of appendicitis give little in the way of symptoms, usually there is no mistaking an attack. There is nausea, vomiting, abdominal pain with tenderness on pressure over the region of the appendix, fever, and an increased number of white blood cells in the blood. Once a definite diagnosis of appendicitis has been made, not a minute should be wasted before operating. Laxatives or sedatives should never be given, as the first only stirs up the bowels and the second obscures the characteristic symptoms and signs necessary for diagnosis.

There is hardly any excuse for death from appendicitis if operation is done immediately after the first definite signs of an attack have appeared. The sooner the operation is done the less chance there is of complications and the more rapid the convalescence.

CHAPTER XXIII.

CANCER.

Cancer is a disease that stands out above all others. Its wide-spread occurrence, its relentless attack and steadily increasing prevalence have given it an evil preëminence. It is a disease that has baffled the attacks of the best doctors and research workers all over the world. Our progress is slow, dishearteningly so, but we have won a few of the preliminary skirmishes.

Cancer is not a new foe, though it seems to be becoming more prevalent now than ever before. The Egyptians knew it well, and the oldest medical literature of India and Persia contain references to the condition. Herodotus records a case of cure of breast cancer, which Democedes operated on twenty-five hundred years ago. Galen, in the second century, worked out a theory of the origin of cancer which held sway for over a thousand years. Four fluids, he taught, controlled the various processes of the body. These four were blood, mucus, yellow bile and black bile. When for any reason the black bile could not escape from the body, wherever it settled and became concentrated cancer developed.

It is rather disheartening to realize that in those far off times the treatment was virtually that of today, though in much cruder form. Extensive cutting out of the tumor and a rim of healthy tissue by knife and cautery was the rule, and many good results were obtained. But a little later, when Harvey's discovery of the circulation of the blood and the rapid scientific progress of the Renaissance exploded Galen's theories, the valuable surgical treatment based on the false premises was swept away in the wreckage.

Instead of knife and cautery, arsenical ointments, freak diets, faith cures and salves were used, all to no avail. From this slough of wild theories and unhealthy speculation we are slowly emerging.

The starting point of our modern views of cancer was given by John Hunter, the great English surgeon, about the time of the revolution. He believed tumor tissue to be nourished just as normal tissue is, and that the same biological laws governed the development of both.

With the development of the microscope a new era opened, one that we trust will ultimately lead us to the cause of this baffling condition. Long and careful study led Virchow to state his so-called cellular hypothesis, that cells can arise only from other cells and not from exuded lymph or blood.

Early in the development of animal or human, the cells formed by the dividing egg cell take on distinct characteristics which divide them into three types. One forms the skin and its structures, as well as the brain and spinal cord. This cell group makes up the ectoderm or "outside skin." Another, the endoderm, forms the lining of the intestinal canal and the structures which develop from it, such as liver and pancreas. The third fills in between, making the bones, muscles, tendons, and so on. Not only does a cell develop only from another cell, but the cells stay true to type. A muscle cell would never produce a skin cell, nor could a skin cell produce a liver cell. That is, not only does a cell arise from another cell, but from another cell of the same sort.

This holds true of cancer cells as well as normal cells of the body. Whatever it may be that starts a cancer to growing, the cancer cells breed true. If it starts from the cornified epithelium that makes the outer layer of the skin, no matter where that cancer grows its cells form cornified epithelium. If it starts from the stomach lining its cells look like those of stomach lining even if they are growing in a lymph node a good ways off.

As happens in any line when a great deal of careful work is done, there are many fine distinctions and precise use of terms. There are a few which it is well to keep in mind. A tumor is an independent new growth of cells, usually of one given type. Tumors may be benign, when they grow but do not invade other tissues. They are called malignant when they invade and destroy normal body tissue. The common name for malignant tumors is cancer.

Cancer spreads in several ways. It extends out at the edges invading the nearby normal tissue. The cells may work their way into the lymph stream and be carried by it to the lymph nodes or other organs. If in an operation a few cells of the cancer are left behind, the tumor will grow again. Wherever a cancer cell is carried and gains a foothold, it multiplies and forms a cancer of the same sort as the original one. These transplanted cancers are called metastases. In most cases, it is the metastases rather than the growth of the original cancer that cause death. Thus a cancer of the breast is rarely fatal in itself, but the various little cancers spread from it like grass fires set from a burning brush-pile do the major part of the damage. They may be in the lungs, the liver, the bone-marrow, practically anywhere in the body. And each one looks the same, under the microscope, as does its parent cancer.

In malignant tumors originating from the connective tissue structures, technically called sarcomas, the spread is usually by way of the blood stream. These also breed true. As a general rule, sarcomas are among the most rapidly fatal types of malignant tumor. Under the microscope the cells can sometimes be seen to be dividing into three, four and even five instead of the usual two, so great is the capacity for growth, far more than could ever occur in any normal tissue.

With these facts in mind, it is simple to pass judgment on the various theories as to the cause of cancer. You probably have seen claims that one or another microbe caused cancer.

So far as we know, a microbe causes the same reaction wherever it invades the body. The germ that causes boils is met by a collection of pus cells whether it invades a hair follicle or gets into an ulcerated tooth. Wherever the tuberculosis germ attacks us, whether lung, bone or brain is the point of assault, the defence is the same, a peculiar kind of white cells, arranged in a characteristic way, and large cells made of several white cells run together. And so it goes with all the germs we know.

It is hard to conceive of a microbe which when it got into the skin made the skin cells grow in a lawless way, but had no effect on the neighboring muscle cells, when sometimes we find the muscle cells rioting in fruitfulness while the adjacent skin cells go placidly along in their accustomed manner.

Moreover, most of the diseases caused by microbes are infectious. There is no proof that cancer is contagious and a great deal of proof that it is not. I have handled a good many hundred freshly removed cancers with bare fingers, but so far I have escaped. I have not even started to worry.

Another objection to the germ theory is that cancer usually occurs in people over thirty-five or forty years of age. It would be a very patient and discriminating germ that would wait for its prospective victim to grow to forty years before making trouble.

Every conceivable microbe or parasite has been blamed, but one by one the theories have fallen before the advance of knowledge. Moreover, tumors do not occur in man alone, but insects, fish, birds and mammals all have them. This would mean that the tumor-forming microbe would have to be able to live equally well in all these different animals, under tremendously varying conditions. No germ we know or dream of could do such things.

Recently Dr. Gye and Mr. Barnard, working in England, announced that they had discovered the cause of cancer and wide publicity was given to their claims. They are working with the so-called Rous chicken sarcoma. This tumor was

discovered in a chicken a number of years ago by Dr. Rous of the Rockefeller Institute. When this tumor was ground up very finely and passed through a special filter that holds back all ordinary germs, the filtered fluid still could cause sarcoma when injected into a chicken.

Dr. Gye believes that a very minute living virus is present in the filtrate. This living virus, however, cannot of itself attack cells and set up tumor growth. It must have the help of some substance or substances found in extracts of tumors. When the virus and tumor extract are injected together, a sarcoma results. When either are injected alone, nothing happens.

The theory is a pretty one. However, there are many objections to it. Chief among them is that no matter what kind of cancer is used to provide the virus-free extract, the resulting tumor is always a Rous sarcoma. Until Dr. Gye's work can be thoroughly tested we are not safe in assuming that the cause of cancer has been discovered. Those men who know most about cancer are somewhat skeptical as to the relation of this virus to cancer.

However, though no spectacular advances in our knowledge of cancer have been made, gradually a considerable body of definitely proved facts has grown up.

There are many cases known where cancer has developed from long-continued irritation, and this undoubtedly may be one of the causes. Thus the irritation of a jagged tooth against the cheek or of a pipe on the lip may, under some circumstances, give rise to a cancer. The effect of exposure to roentgen-rays over long periods, such as the pioneers in roentgen-ray work had to face, results in a slowly-growing but very persistent type of cancer. But chronic irritation in itself is not a cause; something must coöperate with it. Is approaching old age, heredity or something else the necessary factor?

Some forms of cancer apparently develop from cells which

got separated from their normal location as the embryo developed, and then somehow are stimulated to grow.

Practically every possible theory has been advanced, and all have been found wanting in some degree.

While we are waiting for the sure and relatively simple treatment that can come once we know the cause, and better still the prevention that will then be possible, it is important to remember that we have pretty fair tools at hand. If the cancer is found early and thorough surgical removal done as soon as possible, the chances of recovery are very good. But they ebb rapidly as the growth of the tumor goes on. Radium and roentgen-ray are very useful for certain types of skin cancer. Their value, too, depends on prompt application.

Do not wait to be sure a lump or ulcer is cancer, go to your doctor at once. The longer you delay the less chance he has to help you.

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